Posture maintenance at the human elbow joint

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POSTURE MAINTENANCE AT THE HUMAN ELBOW JOINT

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Referent : Dr. E. Otten

On the cover: a light microscopic image of a muscle spindle in the rat soleus (magnification ±500x; kindly provided by dr. R.S.B. Liem).

STELLINGEN

1. Onthouden leidt tot kennis, vergeten tot begrip.

2. Het spreken over 'myotatische *rek*-reflexen' wordt meer gevoed door metrische dan door inhoudelijke overwegingen.

3. Rekkingsoefeningen tijdens het sporten zijn van belang om een verhoogde tonische spieractiviteit te verminderen door het verbreken van het in spierspoelen optredende klitteband-effect (Hagbarth, Hägglund, Nordin and Wallin, 1985).

4. De rusttremor bij de ziekte van Parkinson kan tijdelijk verdwijnen door het passief rekken van de de bij de tremor betrokken spieren.

5. De 'long-latency stretch reflex' is een aangeleerde (pseudo-)reflex, die kan worden versterkt of afgezwakt naar gelang het beoogde effect (Marsden, Merton and Morton, 1981).

6. Het tijdens een lezing gedeeltelijk afdekken van een overheadsheet getuigt van weinig vertrouwen in het vermogen van toehoorders om paralelle informatie op selectieve wijze te verwerken.

7. Veel nuttige informatie verzameld door de zogenaamde basiswetenschappen blijft -ten onrechte- verholen voor de kliniek.

8. Uit het feit dat dit proefschrift, 402 Kbyte groot, gebaseerd is op 200 Mbyte dataregistratie, mag blijken dat het wetenschappelijk proces voornamelijk gekarakteriseerd wordt door een vijfhonderd-voudige datareductie.

9. Bij het werken met de ellebogen geven spierspoelen nauwgezette informatie omtrent de voortgang die is geboekt.

10. Jonge fysiologen zijn ook fysiologen.

11. Het propageren van een hogere leeftijd voor het krijgen van het eerste kind kan een belangrijke bijdrage leveren tot het terugdringen van de overbevolking.

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POSTURE MAINTENANCE AT THE HUMAN ELBOW JOINT

CHAPTER 1

Introduction

1.1. INTRODUCTION

In physiology the maintenance of joint posture is generally thought to arise from the action of myotatic reflexes in the muscles spanning a joint[1]. A myotatic reflex is the abrupt muscle contraction that occurs in response to a brief muscle stretch (for instance, by a tap with a tendon hammer). This reflex cannot be voluntarily suppressed. It is initiated by the excitation of receptor organs (muscle spindles) within the stretched muscle, followed by a volley of actionpotentials traveling up to the spinal cord. In the spinal cord, these actionpotentials cause a direct excitation of the homonymous (of the same muscle) alpha-motoneurones, by means of a monosynaptic projection.

Muscle spindles are highly specialized intramuscular receptor organs, sensitive to muscle length. They contain modified muscle fibres which have maintained their contractile properties. These fibres are called intrafusal fibres, to distinguish them from the so-called extrafusal fibres which are the fibres the muscle is composed of (Lat. fusus = spindle). There are two kinds of intrafusal fibers: the nuclear bag and the nuclear chain fibers. Both kinds of intrafusal fibre have been illustrated on the cover of this thesis.
Nuclear bag fibers are further subdivided into the dynamic bag_1 and the static bag_2 fibers. Both types of nuclear bag fiber are characterized by a central region containing the fiber's nuclei. This part of the fiber is not contractile. On either side of the central region are the so-called polar regions. The polar regions contain actin and myosin filaments and, therefore, have the capacity to contract.
In nuclear chain fibers the nuclei as well as the contractile filaments are found over the full length of the fibre. Several investigations have shown that the nuclear chain fibres need to be subdivided in short and long chain fibers to be distinguished on basis of their histochemistry and innervation (Harker, Jami, Laporte and Petit, 1977; Kucera, 1982).
The muscle spindle is connected with the central nervous system (CNS) by means of afferent (transmitting actionpotentials from the spindle to the CNS) and efferent nerve fibers (from the CNS to the spindle). There are two types of afferents: the primary (or Ia-) and the secondary (or II-) afferents. Both types of afferent provide information on the actual muscle length (the static sensitivity). In addition, the primary afferent also provides information on a change of

[1] This is the so-called feedback control of joint posture, whereas, theoretically, also a feedforward control may exist arising from posture oriented central motor programs.

muscle length (dynamic sensitivity). Apart from these afferents, the muscle spindle receives two types of efferents: the static efferents and the dynamic efferents. These efferent nerve fibres stem from so-called gamma motoneurones within the spinal cord. Whereas the alpha motoneurones innervate extrafusal muscle fibres, the gamma motoneurones innervate the intrafusal muscle fibres. Gamma (or fusi-) motoneurones are subdivided in two types: (1) the static or γ_S fusimotor neurones, which increase the spindle's static sensitivity (and reduce its dynamic sensitivity) and (2) the dynamic or γ_d fusimotor neurones which increase a spindle's dynamic sensitivity. Physiological recordings from muscle spindles while they were simultaneously observed under a microscope (Boyd, 1985), shows that both types of fusimotor neuron innervate different subgroups of intrafusal fibres: the γ_S neurones innervate the static bag_2 and nuclear chain fibres, whereas, the γ_d neurones innervate the dynamic bag_1 fibre.

By its action (muscle contraction in response to muscle stretch) the myotatic reflex seems highly suited for restoring posture once it is perturbed. However, postural mechanisms should not only rigidly maintain a single joint angle; they should also have the capacity to maintain any angle within the limits of joint flexibility. This is called postural plasticity (the ability of a limb to assume and maintain any possible joint angle) and may theoretically require gain modulation (or reflex gating) which permits postural mechanisms to be turned off, when movements are to be executed.

This thesis has developed based on a research proposal containing a working hypothesis on how the central nervous system (CNS), in interaction with its periphery (proprioceptors[2], muscles, etc.), might achieve such postural plasticity. In retrospect, this hypothesis, although rejected at a later stage, proved of major importance throughout the course of this study: it has influenced the way in which the investigation was carried out, as well as, what conclusions were drawn from the experimental results. Therefore, it was thought of interest to repeat part of this early research proposal within the contents of this chapter (paragraph 1.2.). Subsequently, paragraph 1.3. shall briefly describe the subject of the next four chapters as well as how these chapters relate to the questions put forward in paragraph 1.2.

[2] The term proprioceptor designates all receptors that provide information about the internal state of the organism with respect to muscles, tendons and joints. Stimuli to these receptors are traceable to actions of the organism itself, hence the prefix proprio- (Evarts, 1985).

1.2. A WORKING HYPOTHESIS TO INTEGRATE THE FUNCTIONAL PROPERTIES OF MUSCLE SPINDLES INTO AN 'AGONIST-ANTAGONIST' MUSCLE SYSTEM

Around the turn of the century Sherrington (1910) observed that after decerebration[3] in the cat an extreme rigidity of the extensor muscles occurred. He proved that this rigidity could be abolished by cutting the dorsal roots which suggested that the hypertonia of the muscles was due to the activity of spinal afferents. By eliminating all other possible sources of afferent input he was able to prove that proprioceptive activity was essential for the decerebrate rigidity.

Some thirty years later, Pollock and Davis (1931) introduced a new method of decerebration consisting of occlusion of the blood supply to the cerebral cortex. In their preparations, however, rigidity could not be abolished by severing the dorsal roots. They argued that their technique of anaemic decerebration had a less traumatic effect, and that this could explain the differences after sectioning the dorsal roots in their preparations compared to those of Sherrington. Yet, Stella (1944) and later Eldred, Granit and Merton (1953) emphasized that both methods of decerebration differed with respect to the affected brain structures. In the anaemic preparation the anterior part of the cerebellum was also deprived of blood whereas its function was unimpaired in Sherrington's preparations. It was concluded that there are two types of rigidity: 'gamma-rigidity', which is dependent on augmented (due to gamma-fusimotor action) spinal input from muscle spindles; and 'alpha-rigidity' which does not disappear on cutting the dorsal roots.

This is one of the main complications of motor control: there are at least two different systems acting on the tone of a muscle: one system increases tone via the gamma loop; the other acts directly on the alpha-motoneurones themselves. The question how the spindles may contribute in the control of muscle tone has been approached along to basically different lines: the first takes as a pre-assumption that muscle spindles are important for posture control (e.g. Sherrington, 1909), the second is based on the idea that muscle spindles are involved in providing feedback information during the execution of delicate movements (e.g. Merton, 1953). In general, most experiments and hypotheses have been developed along one of these two lines, without paying much attention to their interaction. The present research proposal aims to consider a hypothetical mechanism how spindle afferents and efferents may achieve a fluent transition from posture to movement control.

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[3] Decerebration, as performed by Sherrington, involves the surgical transection of the cat's midbrain. This transection is usually performed in an intercollicular plane, separating the superior from the inferior colliculus.

Formulation of the basic problem

If we consider a simple joint governed by two antagonistic muscles, with its movement confined to only one degree of freedom (Fig. 1.1.), we can rearrange the separate components in a scheme as provided by figure 1.2. The only common output of both muscles is the angle at which the joint is held. Such a simplification can summarize the action of triceps, biceps and brachioradialis on the elbow, as well as the effect of quadriceps and hamstrings on the knee joint.

Figure 1.1.

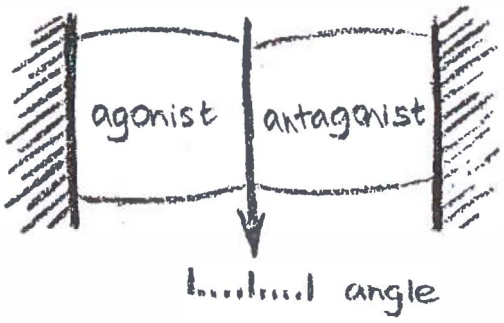

Figure 1.2.

It is assumed that, in figure 1.2., the representation of both muscle groups incorporates their passive elastic properties, otherwise the system would become trivial as the forces exerted by both muscles are identical for any given angle. Therefore, the limb can only be held in a certain position if both muscles are balanced sufficiently to account for the elastic forces driving the limb back to its position of 'iso-elasticity'. Furthermore, any effects of gravity were ignored, assuming that all movements of the limb are within a plane perpendicular to the downwards directed gravitational force.

By incorporating muscle spindles, the alpha and the gamma-motoneurones and their well-known connections, we reach the scheme of figure 1.3 (note that for reasons of simplicity the γ-motoneurones were not further characterized as being static or dynamic). A similar illustration can be found in every basic textbook on physiology.

However, having done so, we are struck by the puzzling conclusion that the basic segmental reflex organization is one of rigidity. Without assuming additional mechanisms, such as yet unmentioned segmental reflex pathways or supraspinal influences, such a system cannot easily

be imagined to be incorporated in any movement. For each movement, passive as well as active[4], will immediately be antagonized by reflex changes in muscle activity.

With respect to supraspinal influences, there are two concepts aiming to explain for this apparent paradox. The first concept assumes that a descending motor command to the agonist motoneurones is always accompanied by an inhibitory command to the antagonist motoneurones in order to cancel the expected effect of the antagonist's stretch reflex. This may either involve a direct or indirect inhibitory pathway (review: Hulliger, 1984). The second

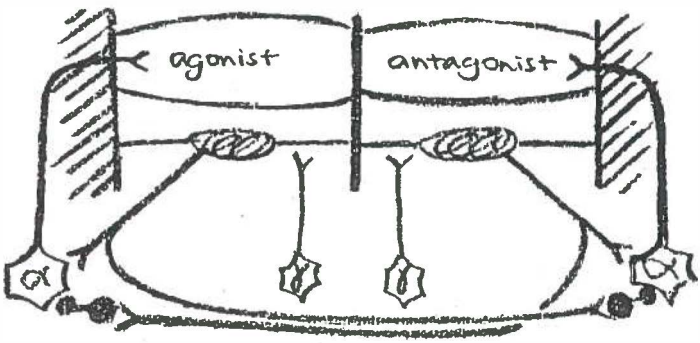

Figure 1.3.

concept assumes that a descending motor command to a muscle's alpha motoneurones is always joined by a simultaneous activation of the muscle's gamma motoneurones (α/γ coactivation; see Vallbo, 1971 and Burke et al., 1979). In this way, the loss of afferent activity expected as a result of muscle shortening may successfully be prevented by an increased spindle sensitivity as result of γ-activation. Similarly, the increase in antagonistic Ia-afferent activity may be prevented by a simultaneous γ-inhibition (α/γ co-silence; see Hagbarth, 1981).

However, both concepts complicate the ideas about motor control. They assume activation of multiple pathways during any voluntary movement. A movement-command must reach the alpha-motoneuron of the agonist muscles whilst simultaneously inhibition should occur of either the antagonist or of the antagonist's stretch reflex activity. Furthermore, whereas these concepts may provide a satisfactory explanation of what happens during an active movement, it is hard to understand what happens during a passive movement, for instance when a physician

[4] The term passive movement is used to designate movements imposed on a subject's limb by means of an external force; this in contrast with so-called active movements, where the movement is induced by muscle activity of the subject him(her)self.

tests a patient's muscle tone. Would these inhibitory pathways also be activated by an imposed movement?

Before accepting that (active or passive) movements are carried out in such a complex way we will consider an alternate solution to the problem of rigidity based on an hypothetical arrangement of muscle spindle afferents and efferents at a segmental level.

A hypothetical solution to the problem of rigidity

A first solution to the problem of segmental rigidity was proposed by Merton (1953). He suggested that the whole problem could be avoided if all voluntary action was carried out by activation of the alpha-motoneurones via the gamma-Ia-reflex loop. He assumed that the signal to the gamma-motoneurones was one of 'desired length' and that alpha motoneurones would adjust their activity until this length was reached and the shortening of the muscle had decreased the Ia-activity. Supporting evidence was provided by Hunt (1951) and Eldred et al. (1953).

Merton's theory of servo control of movement prescribes that gamma activation occurs before alpha-activation. However, Vallbo's experiments on humans (Vallbo, 1971; Vallbo et al., 1979), using microneurographical techniques, proved that it was the other way round and that spindle activation started after the onset of skeletal muscle contraction. This observation has been confirmed in several studies since (e.g. Hagbarth et al. 1975; Burke and Eklund, 1977). These observations, together with the general objection that each voluntary movement, according to the theory of servo control, would suffer a certain delay by first activating the gamma-motoneurones, led to the formulation of the concept of alpha-gamma co-activation. The co-activation (and co-silencing) of alpha and gamma motoneurones (as a neuronal mechanism) can provide a motor system with so-called servo assistance (as control principle; Marsden et al., 1976). By continuously maintaining the muscle spindles 'on tension' it is thought that any deviation of a moving part of the body from its intended trajectory will immediately result in a correcting signal arising from Ia-afferent feedback. For an extensive discussion of these topics see Prochazka and Hulliger (1983) and Hulliger (1984).

However, the concept of α/γ co-activation requires a great deal of co-ordination from supraspinal structures sending down the required patterns for motor activation and inhibition. To escape from such complicated speculations on supraspinal motor control, it was decided to concentrate on spinal mechanisms. Firstly, it is assumed that muscle spindles are only involved

in the maintenance of posture[5] and not in movement control at all. From their connectivity (Fig. 1.3.) it can be seen that spindles are most suitable for stabilizing the angle of a joint.

Accepting the assumption that muscle spindles are only concerned with the maintenance of posture, we can ignore questions concerning the role of muscle spindles in movement control and whether input from muscle spindles reaches consciousness or not, as maintenance of posture is a subconscious and automatic task. The only question we are still facing is how the stretch reflex is inhibited when a movement is made and how it becomes active again once the movement has ceased and a newly acquired position has to be maintained. In other words: how does the system arrange that the stretch reflex can quickly 'thaw' when a movement starts and can quickly 're-freeze' when the movement is completed?

This concept of 'thaw and freeze' bears a strong resemblance with the concept of plasticity first observed and described by Sherrington (1909): in the chronic spinal dog he distinguishes between a 'shortening' and a 'lengthening' reaction. This subdivision is based on the observation that a muscle maintained its new length after it has been stretched or shortened. These responses are also observed in the acute decerebrate cat and seem to be dependent on an intact proprioceptive arc.

Let us reconsider figure 1.3. and propose a hypothetical rearrangement of muscle spindle afferents and efferents to change the system of rigidity of figure 1.3. into a system of plasticity. What are the main requirements such a system should account for?

1. It should be able to fixate or freeze the joint at a certain position whenever it does not participate in a movement. To do so it should be able to cope with perturbations such as heart pulse, muscle tremor and conduction of movements by the rest of the body.
2. This fixation of the joint should be readily canceled when a passive movement is imposed as, for instance, in the case of a physician testing the muscle tone of a patient by flexing and extending the limb. This is an observational rather than a functional requirement.

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[5] The scope of the word 'posture' needs some clarification.
When a particular joint is fixed at an angle the functional role of both muscles governing the joint will be referred to as 'maintenance of posture'. Thus, in this context posture means fixation of the joint and not the wider definition of keeping the individual in an upright position. This definition allows to consider posture maintenance as an automatic and not a voluntary mechanism.

3. During voluntary movement the forces fixating the joint should be readily canceled, preferably without needing complex simultaneous supraspinal mechanisms inhibiting the stretch reflex of the antagonistic muscle.
4. The antagonistic forces fixating the joint should be limited in order to prevent an unnecessarily high build-up of tension.
5. Though not a requirement in its full sense, it seems preferable that all points mentioned above would be met with on a spinal or even a segmental level (being performed automatically).

The first requirement, as we have seen, can be accounted for by the system of figure 1.3.
The second requirement, however, necessitates an inhibitory influence on the stretch reflex (or at least a removal of normal excitatory drive). This inhibitory effect may be accounted for by a negative feedback loop originating from the autogenetic muscle spindle, at least if we wish to satisfy requirement 5 and not allow the inhibition to be accomplished by supraspinal influences. This forms the second assumption.
Thereby, a complex feedback system is created which will theoretically cause the gamma activity to increase when the muscle spindle activity decreases as well as cause the gamma activity to drop when the spindle activity increases. In other words, this system will keep its muscle spindles 'on tension' by 'clamping' the spindles' afferent activity.
However, the described negative feedback loop would immediately conflict with requirement 1. A stretch of the muscle would now initially evoke a reflex twitch but this correcting force will immediately be abolished by the inhibition of the gamma motoneuron by means of the feedback loop.
It is clear that the sensitivity of the negative feedback loop should be restricted to the passive movements described in requirement 2 and at the same time should be unresponsive to the perturbations described in requirement 1. Perturbations distinguish themselves from passive movements by a high velocity (or frequency) and a small amplitude. With their high length sensitivity and high frequency response Ia afferents are most suitable to fulfil requirement 1 and correct for these small and rapid perturbations. The passive movements used by a clinician, in contrast, are of a low velocity and of a relatively large amplitude. Therefore, it is suggested that the proposed negative feedback loop to gamma motoneurones, providing the system with plasticity, derives its activity from muscle spindle II-afferents, which are known to have a low

frequency response and a low length sensitivity (Matthews, 1972). Including this last assumption we can represent the proposed system as displayed in figure 1.4.

The assumption of a negative feedback loop arising from the autogenetic muscle spindles and inhibiting gamma-motoneurones can find support in observations of Hunt (1951) and Eldred et al. (1953). Hunt finds a decrease in firing rate of gamma fibres on stretch of a muscle in the spinal cat, whereas alpha activity increases. Eldred et al. record Ia activity as a measure for gamma discharge rate. During a sustained stretch a sudden fall in Ia discharge is observed that can only be attributed to a fall in gamma activity. The importance of these observations is

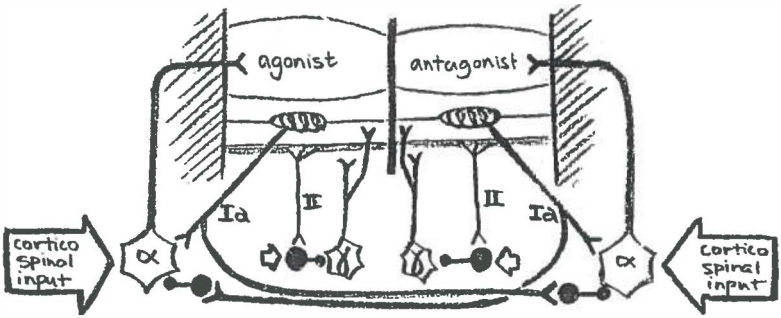

Figure 1.4.

stressed by Granit (1955). He concludes that an inhibitory pathway should be assumed from unknown muscle receptors to the autogenetic gamma motoneurones. He linked this conclusion to reports on plasticity by Sherrington (1909). However, Granit interprets the existence of this pathway as comparable to the effects of Ib tendon afferents on the alpha-motoneurones and considers both together as a double safety mechanism to prevent extremely high muscle tensions.

There are many, more recent, studies of reflex effects on gamma motoneurones, and not all are in favour of the suggested hypothesis. For instance, Appelberg et al. (1983), have convincingly shown that II-afferents have an excitatory, rather than inhibitory, action on gamma motoneurones. Nevertheless, it should always be asked in what kind of preparation a given result is obtained. Where Hunt (1951) demonstrated an inhibition of gamma motoneurones during stretch in the decerebrate preparation, he failed to do so together with Paintal in the spinal preparation (Hunt and Paintal, 1958). In many ways, the spinal as well as decerebrate

preparations are rather unique states of an experimental animal, in which reflex mechanisms may, for instance, be exaggerated which are suppressed in the intact animal (Lundberg, 1982). Results obtained in these preparations cannot always be extrapolated to man.
For human conditions microneurographic experiments seem more relevant. For passive muscles of the forearm it is shown that, when movements are imposed, a Ia-afferent of the shortening muscle silences, whereas that of the lengthening muscle increases its activity (Hulliger et al., 1982). So, in these experiments there seems to be no indication of a decrease in spindle activity during lengthening as reported by Eldred et al. (1953). Still, these results are likely to depend largely on the way subjects have been instructed prior to the experiment. Totally different results may, for instance, be obtained when subjects are asked to contract isometrically or to actively maintain a given joint angle (Hulliger et al., 1982).

Can the model of figure 1.4. satisfy all five requirements? By assuming the selective sensitivity of the negative feedback loop to slow movements of larger amplitude and the insensitivity to movements with high velocity and low amplitude, requirement 1 is secured: During an imposed passive movement the stretched muscle will initially exert a slight opposing tension due to the activation of the stretch reflex. However, this increase in tension will shortly thereafter be antagonized by gamma inhibition, as the stretch surpasses the threshold of the negative feedback loop. Thereby, the muscle gives in to the imposed movement. The muscle spindles of the shortening muscle, on the contrary, are slackened, resulting in both a decreased activity of the Ia- as of the II-afferents. A decrease in II-afferent activity will cause a dis-inhibition of the gamma activity: the gamma system will follow the movement and will try to keep the spindles 'on tension'; a steady Ia discharge is sustained throughout the movement allowing the muscle to shorten actively during the imposed movement.
Concerning requirement 3, during a voluntary movement (induced by contraction of the agonist) the action of the inhibitory feedback loop will readily antagonize the build-up of antagonistic reflex tension resulting from the stretch reflex: again, the increased secondary afferent activity resulting from muscle stretch reduces the spindles' sensitivity by gamma inhibition. For the agonist muscle it is expected that, similar to an externally imposed movement, the drop in II-afferent activity results in a dis-inhibition of gamma motoneurones: the gamma system will actively follow the shortening muscle.
When the movement ceases at a newly acquired angle of the joint the system will automatically stabilize this new posture. The gamma system of the contracting and shortening muscle will

catch up on the movement and at the same time the negative feedback on the gamma motoneurones of the antagonistic side will decrease its activity. The gamma systems on either side of the joint will increase their activity until a sufficient level of Ia mediated alpha excitation is reached to stabilize the joint position. That no unnecessarily high build up of tension on the joint will occur is probably ensured by reciprocal inhibition (or alternatively by the limited gain) of the stretch reflex itself. This accounts for the fourth requirement. The final requirement of segmental automatism is of course fulfilled by the system of figure 1.4.
It is difficult to estimate the quantitative contribution of each pathway to the maintenance of posture as they are all simultaneously active. Computer simulation might prove useful in order to test the flexibility of the system to maintain any given position of the joint. Although the system might not account for all the possible variations in movement and posture, if the majority could be explained on a spinal level, the fine adjustments could arise by supraspinal mechanisms.

1.3. A BRIEF INTRODUCTION TO THE CHAPTERS 2, 3, 4 AND 5

As indicated in paragraph 1.2, it is widely accepted that the stretch reflex is of major importance for postural rigidity (the active maintenance of posture by the central nervous system). The activity of the myotatic stretch reflex stems from the activation of muscle spindle afferents, which are stretch sensitive receptors which present in most skeletal muscles. This study was, therefore, started with an analysis of what is known in literature about the input-output relation of these receptors. There are many reports on this subject which provide a reasonable impression how experimentalists picture the relation between stretch at the muscle spindle poles and the way by which the resulting intrafusal mechanical events elicit muscle spindle discharge (Hasan and Houk, 1975a/b; Boyd et al., 1977; Matthews, 1981; Poppele, 1985; Hunt, 1990). Chapter 2 of this thesis deals with a computer simulation of a muscle spindle. On basis of observed and assumed intrafusal mechanical properties, this model calculates primary afferent activity in response to spindle stretch. This work was carried out in collaboration with dr. E. Otten.
Now a picture had been obtained of muscle spindle physiology, further investigation was aimed to test the validity of the hypothesis proposed in paragraph 1.2. For this, an experimental set-up was designed which allowed to impose perturbations to the arm of a human subject. The

perturbations could be randomized with respect to their amplitude and to their velocity. Muscle activities of the major flexor and extensor muscles of the elbow were recorded as well as the torque required for each perturbation of the arm.

The points of interest in these experiments were: (1) how did the activity of both antagonistic muscles ensure that arm posture was maintained without the occurrence of slow drift or oscillation, and (2) how did changes in muscle activity allow the arm to assume and maintain different arm postures? Chapter 3 deals with the first problem. It is shown how the balanced activation of the antagonistic muscles sets up a 'pit' in the energy landscape in which the arm lies stabilized. Also it is demonstrated how the steepness of this energy pit depends on the level of antagonistic co-contraction. Chapter 4 focuses on the subject of postural plasticity. It is shown that, depending on their amplitude, passive movements of the forearm may induce permanent changes in maintained elbow angle. These changes in elbow angle are brought about by shifts in the tonic activity of the antagonistic muscles.

In order to obtain insight in the origin of observed EMG activities in response to imposed passive movements, we simulated the experimental results by means of a computer model. This model was to contain the hypothetical reflex system as proposed in paragraph 1.2. If such a simulation were successful, this reflex system would still not be proved but would certainly have gained in credibility.

However, at an early stage of the simulation attempts, it appeared that postural plasticity was not likely to arise from the suggested negative feedback loop of secondary afferents to autogenetic gamma motoneurones. In retrospect, this becomes clear if one considers the following argument: on muscle stretch, autogenetic gamma activity needs to be suppressed in order to obtain a lengthening reaction by means of a decrease in primary afferent firing (Fig. 1.4.). This gamma inhibition can indeed be obtained from secondary afferents which are also excited by the muscle stretch. However, the decrease in gamma activity would not only cause a decrease in primary but also in secondary afferent discharge. Thereby, the inhibition by secondary afferents of the autogenetic gamma-motoneurones would yield in importance. In the computer simulation this problem of gamma inhibition followed by gamma dis-inhibition was visualized by a dramatic oscillatory behaviour. Such a problem could only be circumvented if one made extra and ill-founded assumptions, for instance about differential activation of gamma motoneurones or possible non-linearities in spinal reflex transmission. Instead, it was chosen to leave the original hypothesis and search for a new one: postural plasticity was no longer thought

to arise from delicate segmental reflex action, but from characteristic peculiarities in muscle spindle sensitivity as known from experimental studies.

Chapter 5 reports on a model of the human elbow joint. By formulating this model, we became aware of the great number of non-linearities that arise within the postural system formed by the human elbow joint. For instance, the muscle's force-length relationships are non-linear, muscle working arms change with changing joint angles and muscle spindle sensitivity is certainly not a linear function of muscle length. We realized that, comparable to the elbow joint model, the central nervous system (CNS) has to deal with similar sources of non-linearity.

In the model, the processing of sensory information within the CNS was kept simple (only addition and subtraction were allowed). This made it very hard for the model to neutralize non-linearities arising in the periphery. Several hypotheses on the processing of sensory information were investigated by means of model simulation: reciprocal inhibition of the antagonistic alpha motoneurones, Ib-inhibition of autogenetic alpha motoneurones, Renshaw inhibition, etc. The most important problem of the model was postural stability: though the presumed reflexes provided the model with some plasticity, it always suffered from a slow postural drift.

Eventually, after trying various spinal reflex mechanisms in the model of the elbow joint, we had to conclude that the solution for the prevention of slow drift was not to be searched in the processing of sensory information within the CNS. Instead, we became convinced that slow drift was eliminated by a specialization in muscle spindle sensitivity: the occurrence of so-called dynamic stiction. In order to demonstrate this in the elbow model, the muscle spindle model described in chapter 2 had to be revised: fixed cross-bridges within the modeled intrafusal fibres were now allowed to re-establish. Chapter 5 describes how the revision of the spindle model proved essential for the elbow model's postural stability as well as for its plasticity. Again, this work was performed in co-operation with dr. E. Otten.

Concluding, it is hoped that this thesis shows how, throughout this investigation, our views on posture maintenance at the human elbow joint have changed. Where, originally, it was expected that the paradox between postural plasticity and rigidity would be solved at the level of the CNS by a clever way of processing proprioceptive information, we have now reached the conclusion that it are characteristic properties of muscle spindle sensitivity which prevent the system from slow postural drift without impeding the option of plasticity.

CHAPTER 2

A muscle spindle model for primary afferent firing based on a simulation of intrafusal mechanical events

2.1. INTRODUCTION

This study reports on the formulation of a muscle spindle model which simulates primary afferent discharge for various types of stretching and for variable gamma-drive.
Experimental physiologists have formulated several hypotheses how intrafusal events give rise to the characteristic sensitivity of muscle spindles (e.g. Boyd, 1985; Dickson, Gladden, Halliday and Ward, 1989; Matthews, 1981; Proske and Stuart, 1985). We have made it our aim to join these hypotheses into one single model, thereby making it possible to investigate how different hypotheses interact and how this interaction relates to the observed behaviour.
This Introduction will briefly describe the major physiological hypotheses contributing to this study. The Methods section will describe how these hypotheses were combined into one muscle spindle model and how this model was calibrated with respect to its parameters in order to simulate the behaviour of real muscle spindles. The Results section will give an overview of the model's responses when subjected to ramp-and-hold stretches and sinusoidal stretches, respectively. Finally, the Discussion will contain a critical evaluation of the possibilities and limitations of the proposed spindle model. Also, a comparison will be made of the current muscle spindle model with ones previously formulated (Angers, 1965; Chen and Poppele, 1978; Hasan, 1983; Hasan and Houk, 1975; Rudjord, 1970; Windhorst, Schmidt and Meyer-Lohmann, 1976).

Views and hypotheses used for the formulation of the proposed muscle spindle model.
From an early stage on, muscle spindles have been thought to be important in motor control (Boyd, 1985; Matthews, 1981). In consequence, the physiology of these receptors has been studied intensely. Muscle spindles are unique sensory structures as they not only provide information to the central nervous system, by means of the Ia and II-afferents, but also receive

information, by means of the gamma-efferents. This provides the central nervous system with the option to influence the specific sensitivity of the muscle spindle.

Muscle spindles have been shown to be more than simple length sensors. Subjected to ramp-and-hold stretches, for instance, the response of a muscle spindle provides not only information about the actual length of a muscle but also about the velocity of lengthening. The sensitivity of the spindle to the absolute length of a muscle is called 'static sensitivity', whereas the sensitivity to the velocity of stretching is called 'dynamic'.

The specific sensitivity of a muscle spindle is under continuous control from the central nervous system. Matthews (1962) reports that two different types of gamma-motoneurons exist which, on stimulation, may shift the spindle's response either to become more dynamic or to become more static. In consequence, these two types have been called 'dynamic' and 'static' gamma-motoneurons, respectively.

About thirty years ago, the distinction of two types of fusimotor effects on spindle sensitivity was thought to coincide nicely with the morphological subdivision of intrafusal components into nuclear chain and nuclear bag fibers (Jansen and Matthews, 1962). Recent cinematographic studies (Boyd, 1985; Boyd, Gladden, McWilliam and Ward, 1977; Boyd, Gladden and Sard, 1977), however, indicate that, instead of two, three types of intrafusal fiber should be distinguished. Based on these experiments, nuclear bag fibers are now subdivided into two types: dynamic bag_1 and static bag_2 fibers. The dynamic bag_1 fibers are the only ones to receive dynamic fusimotor input whereas the static bag_2 and nuclear chain fibers are exclusively innervated by static fusimotor neurons.

Nevertheless, the morphological subdivision into three types of intrafusal fiber has not yet been followed by a comparable subdivision of muscle spindle sensitivity into three separate types. Consequently, the hypothesis still holds that the distinction of two types of spindle sensitivity, dynamic and static, parallels the subdivision into two subgroups of the three types of intrafusal fiber. It is believed that the bag_1 fiber, innervated by dynamic fusimotor neurons, is responsible for the dynamic spindle sensitivity whereas the bag_2 and nuclear chain fibers, innervated by static fusimotor neurons, mainly contribute to the static spindle sensitivity (Boyd, Gladden, McWilliam and Ward, 1977; Crowe and Matthews, 1964; Dickson, Gladden, Halliday and Ward, 1989; Matthews, 1981).

This subdivision of the intrafusal fibers into two subgroups can also be justified on the basis of ultrastructural observations. An important structural difference between both subgroups is that M-lines in the muscular parts of bag_1-fibers are poorly developed, whereas the M-lines of bag_2

and nuclear chain fibers are identical to those found in extrafusal muscle fibers (Banks, Barker, Harker and Stacey, 1975; Hubbard and Hess, 1967). Matthews (1981) suggests that this difference in ultrastructure gives rise to completely different contractile behaviour of the bag_1-fiber as compared to the bag_2 and nuclear chain fibers. He suggests that the 'creep' of a bag_1-fiber is due to the relative absence of M-lines. Creep is the name Boyd et al. (1977) gave to the observation that the sensory region of a bag_1-fiber during a ramp stretch at first elongates but then, once the stretching has ceased, shortens again to reach its final length. The time course of the bag_1 creep follows the same slow decay as that of the Ia-afferent discharge on the completion of the dynamic phase of stretching (Matthews, 1981).

Not only are structural differences observed between both subgroups of intrafusal fiber, differences in contractile behaviour are described as well. Dickson et al. (1989) report inhomogeneity of sarcomere-lengths in the nuclear bag_1-fiber which is not observed in the bag_2-fiber nor in the nuclear chain fiber. They show that sarcomeres at various locations in a nuclear bag_1-fiber release in groups at different phases of stretching. Thus an elongation of the bag_1-muscle fiber may be divided unequally over different parts of this fiber. It is expected that inhomogeneity in sarcomere-lengths will alter the overall mechanical behaviour of the bag_1-fiber, since inhomogeneity implies differences in stiffness of in series sarcomeres, resulting in uneven interfilamentary velocities. This may change the force velocity characteristics of intrafusal fibres.

Histochemical investigation of the three types of intrafusal fiber showed that the dynamic bag_1-fiber is best described as a slow twitch fiber (type I) whereas the static bag_2-fiber and the nuclear chain fiber are more comparable with fast twitch (type II) extrafusal fibers (Banks, Barker, Harker and Stacey, 1975). This histochemical differentiation agrees well with the observations of Boyd, who reports that the time course of contraction in a bag_1 fiber is more prolonged than in a bag_2 and nuclear chain fiber (Boyd, 1976; 1985)[1].

Several investigators have reported on non-linearity of the muscle spindle response[2]. When Hasan and Houk calibrated their spindle model to observed responses at small amplitudes of

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[1] However, according to Hulliger (1979), this matter is not fully closed as on re-inspection of the data of Boyd, only minimal differences in contraction speed can be seen (see also: Hulliger, 1984).

[2] This phenomenon, which in literature is also referred to as 'gain compression non-linearity', was first described by Matthews and Stein (1969a). Its mechanical origin was demonstrated by Hunt and Wilkinson (1980).

sinusoidal stretching, they found that their model overestimated the sensitivity for larger amplitudes (Hasan and Houk, 1975a/b). Apparently, the sensitivity of muscle spindles to small amplitudes of stretching is higher than that to larger ones.

For sinusoidal stretching from 0 to approximately 100 μm the modulation of the spindle response increases almost linearly with the amplitude of stretching. Therefore, this range of amplitudes has been named 'linear range' (Matthews and Stein, 1969a). It is hypothesized (Hasan and Houk, 1975a/b; Matthews, 1981) that the high sensitivity of muscle spindles for stretch amplitudes within the linear range is caused by stiffness of the polar regions of the bag_1-fiber. Such stiffness is thought to arise due to fixation of cross-bridge regions, a mechanism described for extrafusal fibers (Hill, 1968).

If indeed a number of cross bridge regions in the muscular part of the bag_1 remains attached, then, on stretching, most of the elongation will be transmitted to the sensory region. Consequently, the sensitivity of the muscle spindle increases. Only at stretch amplitudes larger than approximately 0.2 % of the resting muscle length (Hill, 1968) these cross-bridges yield. In that case, part of the stretch is taken up by the muscular part of the fiber and this reduces the sensitivity of the spindle as a whole.

From here on the mechanism by which a bag_1 muscle fiber initially displays a high resistance to elongation, due to its fixed cross-bridge regions, will be called 'stiction'. Stiction has also been shown to cause the so-called initial burst on ramp-and-hold stretches. Just after a stretch starts, the primary afferent of a muscle spindle fires two or three actionpotentials at very short interspike times (Jansen and Matthews, 1962). Several investigations have suggested that this characteristic initial burst can be attributed to the occurrence of stiction (Matthews, 1972; Brown et al., 1969; Proske and Stuart, 1985).

Hasan and Houk (Hasan and Houk, 1975a/b) report on the transition of the state of stiction to the state of cross-bridge yielding. They use ramp-and-hold stretches of approximately 200 μm at extremely low velocity. By means of this technique they demonstrate how the primary discharge initially increases almost linearly but then suddenly drops in firing frequency. Such drops in discharge occur several times during a full stretch. They attribute these drops in discharge to the sudden release of groups of cross-bridges within the intrafusal fiber.

All hypotheses summarized so far share the belief that a great deal of the observations on muscle spindle response can be explained, not by unknown peculiarities of the transducer-process, but by mechanical interaction of intrafusal elements. Differences in effect of

stimulation of either fusimotor neuron, static or dynamic, have been related to differences in contractile behaviour of the intrafusal fibers these motoneurons project to. The initial burst and the linear range, two important characteristics of spindle behaviour, are successfully explained by the occurrence of stiction. The observations on creep of Boyd et al. (1977) show that intrafusal events are in synchrony with the observed patterns of discharge, suggesting a relatively simple transducer process. This is further supported by measurements performed by Poppele (1985) which indicate a linear relation between amount of stretch of the sensory region and Ia afferent firing rate.

The belief that the transducer process is indeed relatively simple, is the main motive behind the formulation of the current muscle spindle model. In this model a mathematical simulation of the mechanical interaction between muscular and sensory part of two intrafusal fibers forms the basis for an explanation of complex muscle spindle behaviour.

2.2. METHODS

Basic construction of the model.

For the construction of the spindle model several simplifications and assumptions have been made.

The model consists of two (sub)models, each of one single intrafusal fiber. The first submodel represents the intrafusal fiber receiving dynamic fusimotor input, the dynamic bag_1. The second submodel was chosen to represent the two types of intrafusal fiber innervated by static fusimotor neurons. Thus, this model combines into one the representation of a static bag_2 and of a nuclear chain fiber. It was decided that the model, aiming at a simulation of two types of muscle spindle sensitivity, would not benefit from the implementation of three different submodels. For reasons of brevity both submodels have been named bag_1 and bag_2, respectively.

Both submodels contain a sensory part (the bag with nuclei around which primary afferents terminate) and a muscular part (the contractile portion of a nuclear bag, containing sarcomeres). Each nuclear bag generates a certain amount of depolarization (transducer process) which depends linearly on the length of each sensory region. By this depolarization, each fiber generates a train of actionpotentials in its branch of the Ia-afferent converging fork. The rate of firing is a linear function of the receptor potential and of its rate of change (Hunt and Ottoson, 1975). A spike is generated each time the elapsed interspike time is in agreement with the

calculated firing rate based on the calculated receptor potential. When arriving at the junction of the afferent branches, a competition occurs between these two actionpotential trains. In the model, the final Ia-discharge is driven by the intrafusal fibre providing the highest discharge rate. (A paper dealing with the nature and cause of this competition between multiple pacemaker sites is in preparation).

The model's mechanics

The main assumption of the proposed model is that the muscle spindle's firing properties depend critically on the mechanical interaction between the muscular and sensory parts of each nuclear bag. This interaction can be modeled by solving the instantaneous force equilibria at the nodal points between both intrafusal parts (Fig. 2.1a.).

The sensory part of each intrafusal fiber is represented by one single elastic unit (Fig. 2.1a.). The mechanical parameters of this unit have been chosen identical for either nuclear bag[3]. The muscular parts of each intrafusal fiber, however, were not identical. They were modeled according to Otten (1987). His muscle fiber model generates forces which depend upon fiber activation, fiber length and contraction velocity, much in the same way as known for real muscle fibers (1938).

In the muscle spindle model the mechanical behaviour of the muscular parts of both nuclear bags were able to differ by means of variations in four different constants.These parameters control the maximum active isometric force (k_1 for bag_1 and k_3 for bag_2), the passive elasticity (k_2 and k_4), the maximum force for an eccentric contraction relative to the maximum isometric force (e_1 and e_2) and the passive damping coefficient (b_1 and b_2, respectively). The dependence of the active force upon gamma discharge is a linear one, scaled with respect to the discharge for maximal effect (100 Hz (Boyd, 1985; Boyd, Gladden, McWilliam and Ward, 1977)). As extra property, not implemented in Otten's model, force enhancement was included in response to stretch. This force enhancement is widely recognized in extrafusal muscle fibers (e.g. Edman, Elzinga and Noble, 1984) and does not depend on sarcomere length nor on fiber activation. In the present spindle model force enhancement was chosen as a fixed percentage of maximum isometric force for either nuclear bag (f_e).

Thus, comprised within the formulation of the model is the assumption that the muscle fiber of a dynamic bag_1 during dynamic stimulation may behave completely different from that of a

[3] The independence between both 'nuclear bags' of the 'muscular-parameters' makes it unnecessary to assume independence of the parameters of the sensory regions too. This would, instead of increasing the flexibility of the model, result in redundancy of the set of parameters.

static bag$_2$ during static fusimotor stimulation. This assumption bears on the structural investigations memorized in the Introduction, which have reported differences in biochemistry,

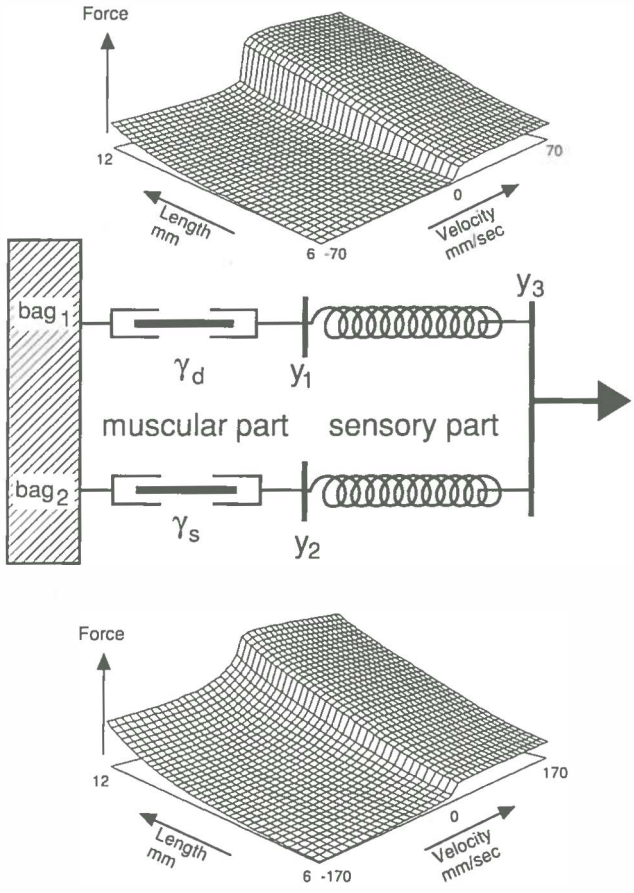

Figure 2.1a.:

Model of a muscle spindle composed of contractile and elastic elements (A). The sensory parts of two different nuclear bags contribute to the firing of one primary afferent. The force-length-velocity relations of the intrafusal fibres of bag1 and bag2 are drawn, showing their different characteristics. The nodal positions (y1 . . y3) are defined as lengths relative to the attachment of the muscular parts at the left hand side.

ultrastructure and contractile behaviour for the dynamic bag_1 compared to the static bag_2 and nuclear chain fibers.
Finally, figure 2.1a. shows that no account is taken of possible effects of intrafusal masses. This also forms an important assumption: mass plays no role of significance for simulation of intrafusal events[4].

The transducer-process and the encoder-process.
Another important assumption of the spindle model, as indicated in the Introduction, is that each nuclear bag contributes to the receptor-potential by means of a relatively simple transducer process. The depolarization generated by each nuclear bag is a linear function of the length of its sensory region. The primary afferent firing rate is a linear function of the receptor potential (compare Hunt and Ottoson, (1975)). However, during the development of this muscle spindle model the calculation of the primary afferent firing rate could not be prevented from becoming slightly more complex. In order to obtain a better simulation of real spindle response, the firing rate was made linearly dependent on the receptor potential and also linearly dependent on the rate of change of the receptor potential (Hunt and Ottoson, 1975). The latter relationship was controlled by the parameter h, the tenth parameter of the muscle spindle model.
For both bags the receptor potential is obtained by multiplication of the length of the sensory zone with a fixed 'length to potential' constant (Appendix C). These constants differ between both bags and are called ltp_1 and ltp_2 (Table 2.2.). In order to obtain the conversion from the receptor potential to firing rate in pps (the encoder process) an extra constant is needed, the potential to rate constant ptr (Table 2.2.). We have found values for ltp_1, ltp_2 and ptr from the following studies: Boyd published experimental data on cat tenuissimus muscle (1976; 1985), which show an elongation of 2 to 8 % of the sensory part of the dynamic bag_1 fiber after dynamic fusimotor stimulation. Also, he found that after dynamic-gamma stimulation the Ia

[4] If the diameter of an intrafusal fibre is 10 μm (Boyd, 1985), then the mass of the whole fibre of length 1 cm can be estimated by taking the product $\pi \cdot (10^{-3} / 2)^2$ $cm^3 = 8 \cdot 10^{-10}$ kg. For ramp-and-hold stretches used under experimental conditions the maximal velocity of stretching was 100 mm/s. Assuming that this velocity was attained 1 ms after the start of the ramp, this indicates an acceleration of 100 m/s^2. Thus, the force required to accelerate the muscle spindle was $F = m \cdot a = 8 \cdot 10^{-8}$ N. This is by far smaller than the force attained by an activated intrafusal muscle fibre. Such a fibre may generate a force of $\pi \cdot (0.01 / 2)^2 \cdot 0.23$ $N/mm^2 = 1.8 \cdot 10^{-5}$ N. The error made by assuming that the mass of the intrafusal fibre does not contribute to its overall mechanical behaviour may therefore be estimated at 0.4 %.

firing rate increased with a maximum of 40 pps. Since the length of the sensory zone of a spindle is approximately known (1.3 mm), we could calculate an estimate of the transition from elongation to firing rate, which turns out to be about 600 pps/mm. For bag_2 an elongation of 12 to 30 % produced an increase in rate of 90 pps (Boyd, 1976; 1985), resulting in about 300

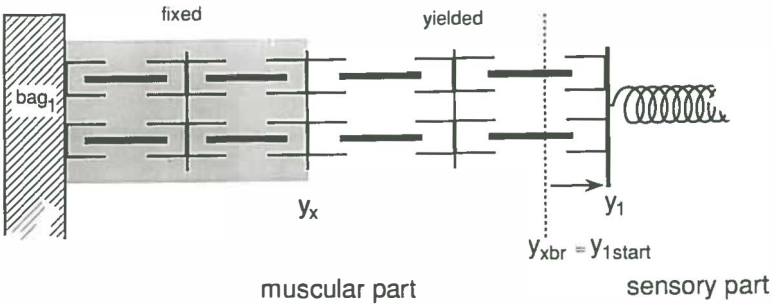

Figure 2.1b.:

Detail of the muscle spindle model symbolizing the model's analog of stiction. For simplicity only four cross-bridge regions have been drawn. In fact, the model contains one hundred of these regions. Two of the cross-bridge regions have yielded and by their lengthening they may have decreased the intrafusal tension. The remaining two are still fixed and behave as a rigid strings, not allowing any elongation.

pps/mm. Hunt and Wilkinson (1980) have made a detailed analysis of the receptor potential in cat tenuissimus spindles in response to sinusoidal stretches. The maximal sensitivity of their muscle spindles amounted 5mV/mm stretching.

This information has been used to discriminate two separate stages in the transformation of sensory zone elongation to primary afferent discharge. Firstly, the transducer process transforms the length signal into a receptor potential. This process is modeled, based on the information mentioned above, by taking ltp_1 = 5mV/mm and ltp_2 = 2.5 mV/mm, (the latter in order to obtain half the sensitivity of the bag_1 fiber for the bag_2 fiber). The second process, the encoder process, describes the transformation of receptor potential to a firing rate. This process was modeled by taking ptr = 120 pps/mV.

Introduction of stiction in the bag_1 fiber

As put forward in the Introduction section the occurrence of stiction in a bag_1 muscle fiber gives rise to the linear range on sinusoidal stretches and to an initial burst on ramp-and-hold stretches.

For this purpose, the description of the muscular part of the bag_1-fiber was expanded with a model-analog of stiction. This was done by introducing a hundred cross-bridge regions[5] in the model. These cross-bridge regions are fixed initially and thereby make the muscular part rigid. If a stretch is simulated in the model, all elongation is initially transmitted to the sensory region, and, due to the resulting stretch of the sensory region, a certain intrafusal force occurs. It is this intrafusal force that can make one of the cross-bridge regions yield when it exceeds a certain breaking force F_x. Thereby, the modeled bag_1 muscle fiber is partially released. Now some of the stretch can be taken up by the muscular part, resulting in a decrease of the intrafusal tension, and (if the breaking force is no longer exceeded) a new state will be reached in which part of the cross bridges is still fixed and part of them has become free (Fig. 2.1b.). In the other case, when the intrafusal force still exceeds the breaking force, another cross-bridge region yields, and this repeatedly so until the intrafusal force is smaller than the breaking force or until all one hundred cross-bridge regions have yielded. Since Proske and Stuart (1985) showed that the initial burst was not restored within several seconds after stretching, cross-bridge regions, once released, were not allowed to become fixed again.
The value for the breaking force F_x was chosen 0.1 FU (FU is an arbitrary force unit, since the spindle model is defined up to a scaling factor, the stiffness of the sensory zone, which, in the model is 1 FU/mm). Chosing this value for the breaking force F_x provided the best match of the model with experimental results on spindle modulation for different amplitudes of stretching (Hulliger, Matthews and Noth, 1977). A formal description of stiction can be found in Appendix B.

Choice of remaining parameter-values.
In order to obtain full definition of the model, a choice still had to be made for the ten parameters, k_1,k_2,k_3, k_4, e_1, e_2, b_1, b_2, h and f_e. Values for these parameters were chosen such that model-responses were obtained which matched as closely as possible with the experimental results on ramp-and-hold stretches of Crowe and Matthews (1964). These authors summarize the response of one muscle spindle to thirty-six ramp-and-hold stretches under six different conditions of fusimotor activity, and for six different velocities of stretching. Of these ramp-and-hold stretches they describe four different features, namely (a) the initial static

[5] The term 'cross-bridge region' should be taken to designate a group of sarcomeres, both in series as well as in parallel, displaying short-range elasticity by the formation of stable actin-myosin bonds.

discharge, (b) the mean discharge during the active phase of stretching, (c) the peak discharge and (d) the dynamic index.

The values for the parameters were obtained by making use of a computerized search procedure based on the polytope method first introduced by Nelder and Mead (1965). This method is useful when a local minimum is to be found of a function that depends on a large number of variables and of which the first (mathematical) derivatives are hard to obtain (as is the case in the present model).

The values for the parameters obtained after optimization are summarized in Table 2.1. Those values that are in arbitrary units (FU) can be multiplied by a constant, without changing the model output.

Once the values for the parameters were found the model was fully defined. The mechanical behaviour of the two muscular parts of both intrafusal fibers has been illustrated in figure 2.1a. by plotting the relation between exerted force versus fiber length and contraction velocity for maximal gamma activation.

Representation of response characteristics of the muscle spindle model.

Once the full set of parameters was defined by the best fit of experimental data of Crowe and Matthews (1964), the model was tested under various conditions and the outcomes were compared with experimental data found in muscle spindle literature. For this purpose two computer-programs were developed, one for ramp-and-hold stretches, the other for sinusoidal stretches.

The muscle spindle model is formulated in terms of spindle-length. Amplitudes of stretching in literature, however, are always with respect to the whole muscle. It was assumed therefore, that any length change of the whole muscle was transmitted completely to the muscle fibers, and proportionally to the muscle spindle. All experimental data used for evaluation of the model response were obtained from the soleus muscle of the cat. This muscle has a fiber length of 42.6 mm (Poliacu-Prosé, 1985). Muscle spindles in cat have a length of almost 10 mm (Boyd, 1976). Therefore, when the soleus muscle is stretched, its muscle spindles will be stretched with approximately 25 % of the muscle elongation.

For comparison of the outcomes of the muscle spindle model with experimental data, definitions will be used as in literature. Thus, for ramp-and-hold stretches for instance, the 'dynamic index' was calculated by taking the difference between the rate of impulses per second (pps) at the end of a stretch and 0.5 s thereafter.

For sinusoidal stretches the model responses were gathered in 24 bins, in phase with the input signal (e.g. Goodwin et al., 1975). Subsequently, the obtained average firing frequencies were fitted with a sine by making use of Fourier-analysis. The frequency of this sine was kept the same as the frequency of sinusoidal stretch. Therefore, we calculated the combination of

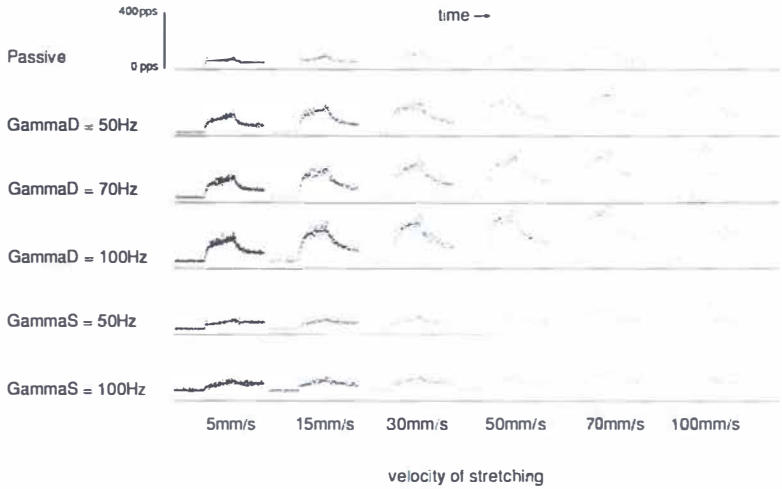

Figure 2.2.:

The response of the spindle model to ramp-and-hold stretches of variable velocity under different conditions of fusimotor drive. The dots represent individual spikes with their reciprocal interspike times simulated by the model. The thin lines denote the base-lines. The bar at the upper left shows the calibration for all traces (0 to 400 pulses per second). The time axes have been scaled with the duration of the ramps. The total amplitude of stretching was 6 mm. for all velocities and for all fusimotor conditions.

amplitude, phase and offset which provided the best fit to the response curve. Modulation (in pps) was defined as the amplitude of the fitted sine. Sensitivity (in pps/mm) was defined as the amplitude of the fitted sine divided by the amplitude of the sinusoidal stretch.

Implementation of the model.

All software was developed within our laboratory and was run on an Apple Macintosh IIx computer. The programs were written in Pascal and used the mathematical co-processor.
All programs are available on request.

2.3. RESULTS

Ramp-and-hold stretches.

As described in the Methods section, those values for the mechanical parameters of the model were chosen which gave the best match to experimental data on ramp-and-hold stretches.

The responses of the spindle model under the conditions used for optimization are shown in figure 2.2. (note that, for reasons of comparison, the time-axes for the different velocities of stretching are adjusted). As can be seen from figure 2.2., the behaviour of the model under

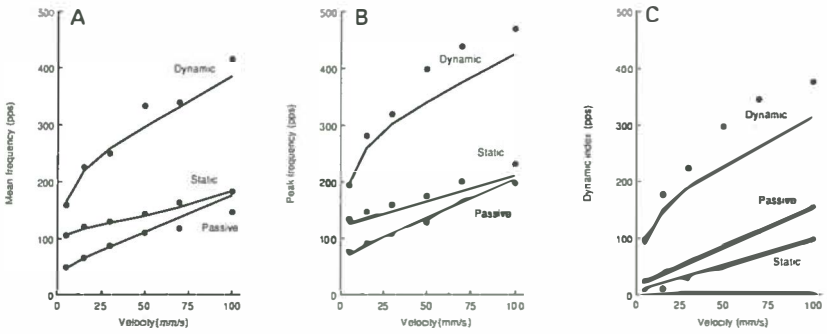

Figure 2.3.:

Experimental results and model simulation for ramp-and-hold stretches.
The dots are extracted from the experimental data obtained by Matthews and Crowe (1964; with permission of the authors). The thick lines represent the model's response. Responses for static and dynamic fusimotor stimulation have each been obtained at a frequency of 100 Hz.
Mean frequency (A): the mean of the firing frequency over the period of active stretching. The peak frequency (B): the maximal response to the ramp-and-hold stretch at the end of the stretching phase. The dynamic index (C): the difference between peak response and discharge 0.5 s after the stretch has ceased.

various regimes of fusimotor drive is in agreement with general physiological theory (Bessou and Pages, 1975; Crowe and Matthews, 1964; Colomo, Lombardi and Piazzesi, 1986): dynamic fusimotor activity makes the spindle velocity-sensitive, static fusimotor activity makes the muscle spindle length-sensitive. Gaussian noise was added to the firing rates with a standard deviation of 5% of the rates to simulate observed noise in spindle experiments. (Matthews and Stein, 1969b).

Plots were made of three of the four characteristic features of spindle output (Crowe and Matthews, 1964) compared with the model's response under identical conditions. Figure 2.3. gives a selection of experimental data and of the model's responses for passive circumstances, for 100 Hz static and 100 Hz dynamic fusimotor stimulation.

As can be seen in figure 2.2., the initial static discharge of the model (before the onset of each ramp), shows some increase under dynamic as well as under static fusimotor stimulation. Static fusimotor activity results in a slightly more prominent rise of the initial firing frequency.

Greater differences in effect between both types of fusimotor drive occur during the dynamic phase of stretching. That even at low velocity the spindle model still displays some dynamic activity (as real muscle spindles do) should partially be attributed to the implementation of force enhancement, giving rise to a steep increase in intrafusal force as soon as the spindle starts stretching, and partially to the force-velocity characteristics of the intrafusal muscular parts (the effects if both mechanisms is represented by the plots of figure 2.1a.).

The model's responses in figure 2.2. are more angular than in simple models consisting of linear springs and dampers. This is caused by the non-linearity of the force-velocity characteristics of the model's muscular parts (Appendix A). At low velocities of stretching the model's muscle fibers increase their pulling force drastically at the onset of the ramp but this increase saturates once the muscle fibers reach higher stretch velocities, thereby making the response curves more angular.

When the active phase of stretching comes to an end, the sensory regions stop elongating. Instead they slowly shorten, while the muscular parts still become longer. The model makes use of the sudden change from lengthening to shortening of the sensory region of the dynamic bag_1 fiber in order to mimic the instantaneous drop in discharge seen in responses of real muscle spindles (Crowe and Matthews, 1964a/b; Matthews, 1962). This effect is simulated by the encoder process as it is not only dependent on the generator potential itself, but also on its rate of change. therefore, the sensitivity of the encoder process shifts from high to low when the rate of change of the generator potential turns from positive to negative.

As illustrated in figure 2.1a., the bag_2 fiber is stiffer than the bag_1 fiber, whereas the latter has a stronger force-velocity effect than the former. This results in the ability of the model to behave either more as a length transducer at static fusimotor stimulation and more as a velocity transducer at dynamic fusimotor stimulation. The increased static discharge during static fusimotor stimulation obscures the still present dynamic response of the bag_1 fiber due to the

competition between the action potential trains of both nuclear bags. This effect results in the lowering of the dynamic index during static fusimotor activity (see also Fig. 2.3c.).
The plots of mean, peak and dynamic index (Figs. 2.3a/b/c.) show that model's responses are in good agreement with the experimental data of Crowe and Matthews (1964).

Sinusoidal stretching.
Since the model was calibrated for relatively large (6 mm soleus stretch) ramp-and-hold stretches, testing the response of the model to small amplitudes of sinusoidal stretching could reveal possible limitations.
Traditionally, the behaviour of muscle spindles during sinusoidal stretching is presented in plots of specific primary response characteristics (such as modulation, phase and sensitivity) to the amplitude or to the frequency of stretching. For our model we used the same testing conditions as used for the analysis of real muscle spindles.
Figures 2.4a. and 2.4b. are plots of the depth of modulation and the phase of real spindle responses as function of sinusoidal stretching of the cat soleus muscle at a frequency of 1 Hz. Responses are plotted for passive circumstances as well as for both conditions of fusimotor drive (Hulliger, Matthews and Noth, 1977). In these figures the solid lines represent the responses provided by the spindle model.
Figure 2.4a. shows that proportionally, the model provides a good approximation of the modulation of real muscle spindles in response to sinusoidal stretching at 1 Hz. Note however, that the model modulations are off by a factor of two. This is due to the fact that the model parameters have been optimized for ramp responses in which stretch velocities are much higher.
As was already mentioned in the Introduction section, a modulation-versus-amplitude plot contains a so-called linear range. This linear range is most marked for passive circumstances and occurs at sinusoidal stretches up to approximately 100 mm of soleus stretch. For dynamic fusimotor stimulation no clear linear range can be distinguished. On the contrary, dynamic fusimotor stimulation even seems to lower the spindle's sensitivity for smaller amplitudes. This effect causes the response of the dynamically activated ending to become smaller than that of the passive spindle, giving rise to the characteristic cross-over amplitude (Hulliger, Matthews and Noth, 1977). At the cross-over amplitude the response of the dynamically activated ending for the first time surpasses that of the passive ending.
Since stiction was included into the muscle spindle model (Fig. 2.1a., Appendix B), linear range (especially for the passive ending) and cross-over amplitude were successfully simulated

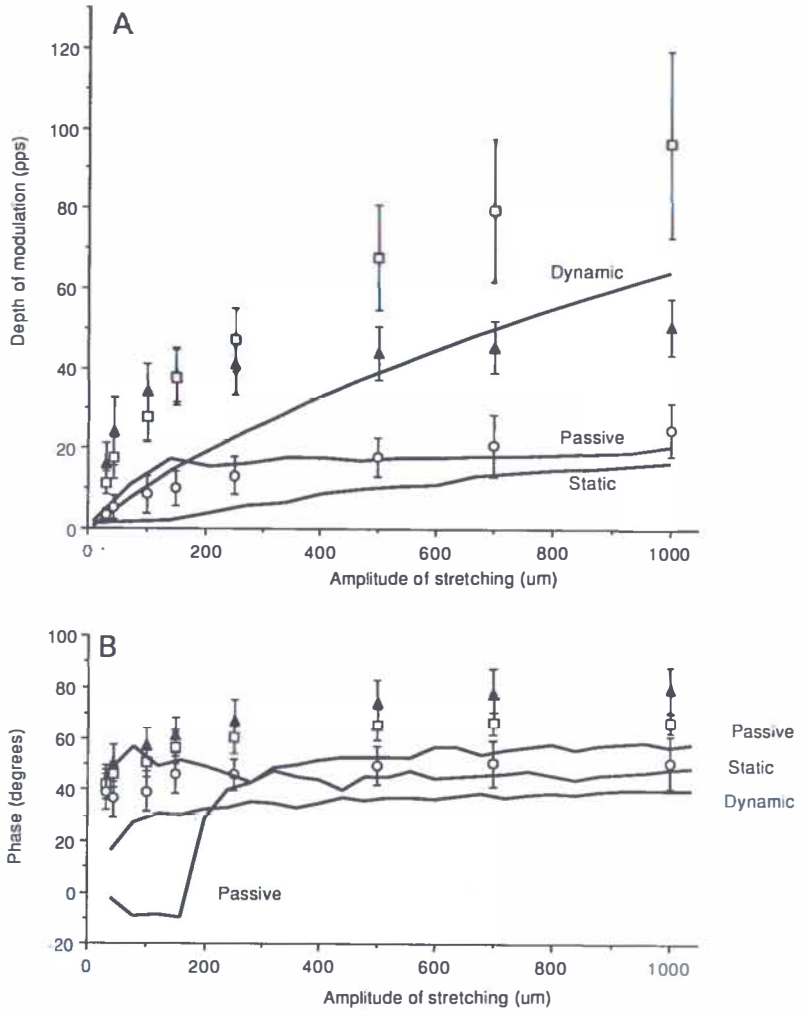

Figure 2.4.:

Experimental results (Hulliger et al., 1977; with permission of the authors) and model simulation for sinusoidal stretches of varying amplitude.
Modulation (A) and Phase (B) versus amplitude plots for sinusoidal stretching at a frequency of stretching of 1 Hz. Meaning of the symbols for experimental data: filled triangles passive, open rectangles dynamic and open circles static fusimotor conditions; vertical bars denote ± 1 Standard Error of the Mean. The continuous lines represent the response characteristics of the spindle model.

(Fig. 2.4a.). Nevertheless, the linear range of the model extended up to approximately 130 mm. For small amplitudes within the linear range, due to the fixation of cross-bridges, all length change was transmitted directly to the sensory region of the bag_1-fiber. Though the transducer process was perfectly linear, the encoder process was non-linear as it depended both on the generator potential as well as on its rate of change. Thereby, the encoder process caused the model's response within the linear range to deviate slightly from perfect linearity.

The cross-over amplitude of the spindle model occurred at approximately 170 mm of soleus stretch (Fig. 2.4a.). During dynamic fusimotor stimulation the modelled force-velocity effects gained in importance. Therefore, the release of one cross-bridge region reduced the intrafusal tension only slightly. Consequently, under dynamic fusimotor stimulation, more cross-bridge regions were released at a given amplitude than under passive circumstances. This reduced the spindle-sensitivity during dynamic stimulation as compared with passive circumstances. Under passive circumstances the fiber can still be in its linear range, where under dynamic gamma drive all cross-bridge regions would have yielded.

The fact that dynamic fusimotor stimulation gave less rise to stiction-effects is in agreement with the assumed mechanism by which stiction occurs. No short range elasticity can be expected when all crossbridges are continuously recycling due to muscle fiber activation.

With regard to the phase characteristics, the model provides a somewhat less favourable simulation. Figure 2.4b. shows the phase-response of spindle output (Hulliger, Matthews and Noth, 1977), as well as of the model. A positive phase with respect to the input signal denotes phase-advance, a negative phase-lag. The phase-response is plotted as a function of the amplitude of stretching.

The data of Hulliger et al. (1977) show that the phase of the primary response is positive over the full range of amplitudes tested. Also these data show that the phase remains largely unaffected by variation of the amplitude of stretching, nor is it dramatically changed by varying fusimotor stimulation.

The model's response falls below the ranges provided by the experimental data for dynamic and passive conditions. This difference suggests that either the bag_1 intrafusal fiber may be more viscous than the modeled one or the transducer is not linear. For passive fusimotor conditions the model's response displays a close to zero phase advance below 160 mm of stretch amplitude, which is in the linear range. The fact that the experiments show otherwise, suggests that intrafusal muscle fibers have visco-elastic properties during stiction, which is not

implemented in the model. The phase under static conditions is in agreement with the experimental results.

With respect to the spindle's frequency response, some discrepancies were seen between model and experimental results. By comparing the experimental data (Goodwin, Hulliger and Matthews, 1975; Fig. 2.5a.) with the results produced by the model (Fig. 2.5b.), some limitations of the spindle model were revealed.

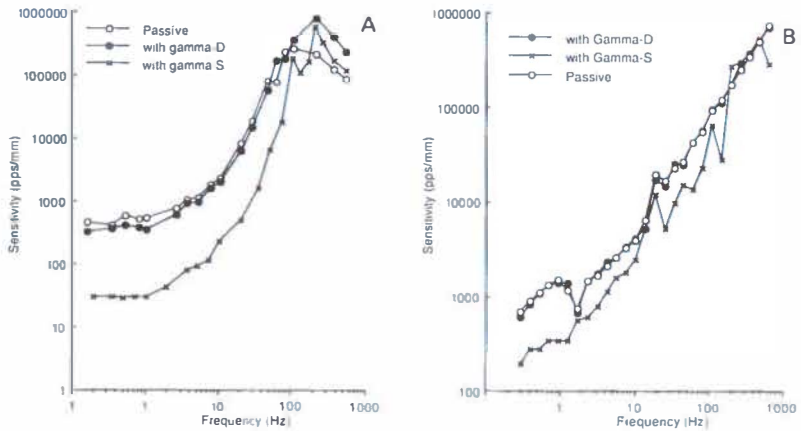

Figure 2.5.:

Bode plots of the gain at different frequencies. A: Replot of the experimental results of Goodwin et al. (1975; with permission of the authors). These results have been obtained with stretches of various amplitude within the linear range but at various amplitudes (see text). B: The response of the muscle spindle model obtained at a amplitudes of stretching of 2 to 40 mm soleus stretch. Although the amplitudes of stretch varied, they all fell within the linear range.

The experimental results were obtained by analyzing stretches within the linear range. However, the exact amplitude of stretching used for this particular plot is not reported by the authors. We were only able to retrieve the range of stretch amplitudes (0.05 to nearly 1000 micrometer (Goodwin, Hulliger and Matthews, 1975)) . For the model we employed a range of 2 to 40 micrometer amplitude, decreasing the amplitude with increasing frequency. Similar to the experimental study, 24 bins per sinusoidal cycle were defined to collect the model's spikes. At each frequency of sinusoidal stretching we chose the mean length of the spindle model such

as to produce the highest attainable sensitivity. A minimum of 500 spikes was collected by using a sufficient number of cycles. The capricious shape of the response plots is largely due to unevenness in the filling of bins. The carrier frequency was roughly 30 pps for every simulation.

2.4. DISCUSSION

Formulation of the muscle spindle model

When we formulated the spindle model we started from the point of view that a great deal of the reactions of muscle spindles to various types of stretching can be explained on the basis of intrafusal mechanical events. Nevertheless, in the course of development of the current model we have been forced to introduce non-mechanical factors also, in order to simulate several of the characteristic non-linearities of muscle spindle response. This was disappointing as non-mechanical factors are often ill-defined and not readily translated into mathematical formulas.

The major part of this Discussion will deal with the characteristic features of spindle response which have been successfully simulated by the model. We will discuss what assumptions were necessary to obtain the requested behaviour. Also, we will focus on the occasions where the muscle spindle fails to provide a proper response. We will indicate what are the most likely causes for discrepancies between experimental results and model responses. Finally, the last part of the Discussion will compare the current model with previous ones and the merits as well as limitations of the current spindle model will be discussed. Several questions will be formulated which may direct future physiological investigations.

A comparison between real muscle spindles and the current spindle model: Ramp-and-hold stretches.

The model simulates the spindle's response to ramp-and-hold stretches with considerable accuracy (Figs. 2.3a/b/c.). The fact that peak frequency and dynamic index at high velocities of stretching stays behind with respect to the experimental data for 100 Hz dynamic fusimotor stimulation, has to do with the fact that the model also needed to simulate the behaviour for dynamic fusimotor drives less than maximal (see Fig. 2.2.). The model, simulating the response of an individual muscle spindle over a wide range of fusimotor conditions and of stretching velocities, behaves satisfactory also with respect to the shape of single ramp response curves (Fig. 2.2.).

A mixed sensitivity to length and elongation was obtained by joining in the model the contributions of two submodels each of one intra-fusal fiber. These two submodels, simulating a dynamic bag_1 and a static bag_2 fiber, received separate innervation and were strictly independent with respect to their mechanical behaviour[6].

After computer optimization, one of both fibers, the dynamic bag_1, has become responsible for the dynamic component in the final Ia-discharge, the other, the static bag_2 (and nuclear chain fiber: see Methods), is responsible for the static component. It is not very likely that in real spindles both tasks are so perfectly separated over the two groups of intrafusal fiber. Nevertheless, the strict separation does allow optimal control over the model's response, shifting its sensitivity from more static to more dynamic.

The values found after computer optimization for the various parameters give an indication what exactly the mechanical differences are between both groups of intrafusal fiber. It proved beneficial to the model's behaviour that (Angers, 1965) dynamic fusimotor stimulation mainly increased the force-velocity effect (viscosity) of the bag_1 muscular part and that (Banks, Barker, Harker and Stacey, 1975) static fusimotor stimulation increased the force-length effect (elasticity) of the muscular part of the static bag_2 fiber, with little effect on its damper characteristics (Fig. 2.1a.). These differences in contractile behaviour under fusimotor activity are in agreement with cinematographic observations on muscle spindles (Boyd, 1976; 1985; Boyd, Gladden, McWilliam and Ward, 1977) which show that on gamma-stimulation the dynamic bag_1 muscle fiber barely shortens, whereas its static counterpart reduces its length markedly[7]. Furthermore, after stimulation the dynamic bag_1 seems to increase its resistance against elongation. This viscous behaviour is thought to be most marked in the polar regions of the nuclear bag_1 fiber (Matthews, 1981).

That, after optimization, the parameters of the muscular parts of both bags are so different is a strong indication that the contractile properties of both nuclear bags need to be quite different in order to explain for the separate control of sensitivity to length and elongation in muscle spindles. Especially the force level at which the bag_1 fiber stabilizes at high stretch velocities compared to the isometric case is very high compared to extrafusal fibers. Extrafusal fibers have

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[6] This assumption may prove only partially true, as cinematographic studies have shown that occasionally contraction of a nuclear bag_1 fibre may unload nuclear chain fibres (Boyd et al. 1976).

[7] This of course is an intuitive argument. So far nobody has been able to actually measure the viscosity of intrafusal fibres.

been reported to achieve force levels of 1.4 to 2.0 times isometric active force (Colomo, Lambardi and Piazzesi, 1986). The intrafusal fiber of the bag_1, however, required a force level of 4.5 times isometric active force. For the bag_2-fiber a value of 1.4 was obtained after optimization which did fall within the range of experimental data on extrafusal properties. The question remains whether contractile differences are due to differences in ultrastructure (Matthews, 1981) or to for instance differences in cross-bridge dynamics.

During the dynamic phase of a ramp-and-hold stretch the primary afferent discharge, after an initial rise, usually increases proportionally with increasing length (Houk, Rymer and Crago, 1981). The spindle model displays similar behaviour in its individual responses on ramp-and-hold stretches, especially for low velocities of stretching (Fig. 2.2.). This effect should be attributed to the implementation of force enhancement as well as to the non-linearity of the force-velocity curves of the model's muscular parts. While the velocity is still low, the resistance of the model's muscular parts is relatively high and a large proportion of the stretch is transmitted to the sensory region. On increasing velocities, however, the viscous resistance saturates and the model's muscular parts take up a larger proportion of the stretch. The dynamic responses become slightly angular which is in agreement with observations on real muscle spindles.

Another striking observation on the effect of fusimotor activity on muscle spindle sensitivity is one reported by Crowe and Matthews (1964). They reported that during static fusimotor stimulation the dynamic index of the spindle response becomes smaller. This observation provides information how the contribution of the model's static bag_2 fiber should be combined with that of the dynamic bag_1 fiber in order to form one final Ia discharge rate:

The responses obtained for passive circumstances are the minimal activities of the muscle spindle which will always be present. Static fusimotor stimulation can only decrease the dynamic index if one assumes that the bag_1 and bag_2-fibers compete in their influence on the final primary afferent discharge[8]. For this reason the contributions of both nuclear bags could not be combined by means of a simple addition. Instead both contributions were combined by taking the strongest discharge of the two. To avoid discontinuities, this competition was simulated with a formula (Appendix C). This allowed one fiber to take over from the other, when the contribution of the first was larger than that of the latter. The dynamic index present

[8] Evidence for such competition between multiple pacemaker sites is reported by Hulliger and Noth (1979).

under passive circumstances mainly stems from the bag_1-fiber. On static fusimotor activity the static background discharge becomes elevated. Consequently, the contribution of the bag_1 (which does not change on static stimulation) 'drowns' in the increased background discharge of the static bag_2.

Close inspection of spindle responses during ramp-and-hold stretches (e.g. Crowe and Matthews, 1964a/b; Matthews, 1962) indicated that at the end of stretching two phases of decay in firing rate may be distinguished: a first and instantaneous decay (sometimes with undershoot) followed by a second decay characterized by a slow time-constant. The slow decay of firing can easily be explained on basis of the observed intrafusal creep (Boyd, Gladden and Ward, 1977), as the time-constants are quite similar. The sudden fall in discharge occurring immediately after cessation of the active phase of stretching is produced by implementation of the effect described by Hunt and Ottoson (1975). These investigators simultaneously recorded intrafusal tension and generator potential of isolated muscle spindles. They demonstrated that changes may occur in generator potential which are not present in the tension recordings. Directly after a ramp-and-hold stretch they observed a phase of relative hyperpolarization which may be held responsible for the rapid decay of firing at the end of a ramp-and-hold stretch. Also these authors compared the relation between generator potential and spindle discharge. When the spindle was kept at a constant length they showed that it was possible to define a linear relationship between generator potential and resulting firing rate. During a ramp-and-hold stretch, however, they showed that the actual firing of the spindle exceeded the firing expected on the base of the measured generator potential. Apparently, the encoding process itself contributed to the dynamic response of the muscle spindle. Actual firing rates exceeded those calculated on basis of the generator potential with a factor of three (Hunt and Ottoson, 1975).

In the model during the hold phase, the sensory zone is allowed to shorten, the length change and the change in generator potential are reversed and, consequently, a sharp decline in firing rate is produced.

Concluding, non-linear properties of the model's transducer and encoder process are essential for an accurate simulation of real muscle spindle behaviour. Clearly, the model's mechanics form the basis of the model but do not suffice alone.

A comparison between real muscle spindles and the current spindle model: Sinusoidal stretches.

Model outcomes to sinusoidal stretches were compared with the experimental data of Hulliger et al. (1977). These data contain measurements on the modulation of the spindle response for

different amplitudes of stretching at a fixed frequency of 1 Hz. It can be calculated that the maximal velocity of stretching used during these experiments did not exceed 6 mm/s soleus stretch[9]. Of course, muscle spindle behaviour to ramp-and-hold stretches should to some extent be reflected in its behaviour during sinusoidal stretching. It was expected that the model would fit responses to sinusoidal stretching at 1 Hz as well as ramp-and-hold stretches at approximately 6 mm/s. Indeed, the model behaved very well for sinusoidal stretches, nevertheless, not without a suitable choice for the breaking force F_x.

Comparable to the decrease in dynamic index for ramp-and-hold stretches, static fusimotor activity decreases the primary afferent modulation in response to sinusoidal stretching (Fig. 2.4a.). As discussed above, this observation indicates that the contribution of both nuclear bags cannot be joined by simple addition: some kind of competition is expected to occur between the contributions of the various intrafusal fibers. If both bags produce action potential trains in either feeding branch of the Ia-afferent axon, the fusion of these trains is always dominated by the most active bag. In this way a competition arises between both intrafusal bags. In the model this is simulated by taking the highest firing frequency of either bag as the Ia-afferent firing rate. Important phenomena such as 'linear range' and 'cross-over amplitude' have been simulated by the model through implementation of an analog of stiction. This short range elasticity was introduced within the muscular part of the nuclear bag_1-fiber.

Under passive fusimotor conditions the main contribution to sinusoidal stretching is derived from the dynamic bag_1-fiber. Within the linear range the muscular part of this fiber behaves as a perfectly rigid string. Consequently, all elongation is directly transmitted to the sensory region. No phase advance is possible in this situation. In real spindles, this phase advance does occur and may stem from the fact that fixed cross-bridges do not behave as perfectly rigid strings. In fact, fixed cross-bridges do allow a limited amount of stretching (Hill, 1968). The repetitive stretch and let go of fixed cross-bridges may result in some kind of viscosity, which in its turn may be responsible for the observed phase advance of Ia-afferent firing in response to sinusoidal stretching within the linear range (Hunt and Wilkinson, 1980)[10].

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9 At sinusoidal stretching the maximal velocity reached in the experiments of Hulliger et al. is calculated by taking $2 \cdot p \cdot$ frequency $\cdot$ amplitude. The frequency used was 1 Hz, the maximal amplitude of stretching 1 mm. Thus the maximal velocity used was 6.3 mm/s at the level of the soleus.

10 An alternative explanation may lie in the recent evidence that even in passive intrafusal muscle fibers cross-bridges undergo a slow cycle of breakage and reattachment (Baumann and Hulliger, 1991)

Furthermore, concerning the model's phase versus amplitude characteristics, Goodwin et al. (1975) have already pointed out that the measurement of phase from cycle histograms of impulse activity is not completely unbiased. Several factors may impair correct measurements of the phase. Artificial phase shifts may be measured due to lagging of the stretch apparatus, due to the fact that the wave of movement is transmitted with a finite velocity along the muscle or, due to conduction times over the nerve fibers to the point at which the action potentials are recorded from. On base of these considerations, Goodwin et al. do not pay much attention to the absolute values of the phase (1975). The occurrence of artificial phase shifts can, however, be expected mainly at high velocities of stretching. Therefore, the observed misfit between model and experimental results for low amplitudes of stretching cannot simply be ignored. We chose to conclude that the model gives an underestimate of the phase advance of real muscle spindles.

Bode plots of spindle sensitivity in relation to the frequency of stretching reveal characteristics of the model. Goodwin et al. (1975) report an extremely high gain (or sensitivity) of the spindle to sinusoidal stretches within the linear range (Fig. 2.5a.). According to these authors, the sensitivity ranges from approximately 30 to almost 1 x 10^6 pps/mm. Within the linear range, the muscle spindle model behaves well, except for the static fusimotor fusimotor condition, in which the values are approximately ten-fold too high.

Hunt and Wilkinson (1980) compared the generator potentials of primary endings (after application of tetrodotoxin) with the primary discharge beforehand. Comparable to the observations for ramp-and-hold stretches (Hunt and Ottoson, 1975) these authors showed that intrafusal tension and generator potential may occasionally change independently. Also they found that on increasing frequencies of stretching (beyond 20-30 Hz) the modulation of the generator potential decreased while in the same range Goodwin et al. (1975) found that the modulation of Ia-discharge showed a steep incline (Fig. 2.5a.). Again this suggests that non-linearities may occur at the level of the transducer as well as the encoder process.

Part of an explanation for the high sensitivity of spindles is based on a careful study of the methods used for experimental analyses. Goodwin et al. (1975) recorded actionpotentials and sampled them in 24 bins which were in phase with the input signal of sinusoidal stretching. Recording times were reported to be in the order of ten seconds so that a sufficient number of sines was sampled. After sampling of the actionpotentials a leastsquare sine-fit was performed on the calculated discharges per bin. This fitted sine provided amplitude (and modulation) as well as phase of the spindle response.

For the lower frequencies of stretching it is clear that the spindle response is accurately represented by this way of analysis. However for the higher frequencies of stretching (up to 500 Hz) action potentials tend to cluster in bins covering only part of the sine wave, so that the fitted sine through the bins tends to have a higher amplitude than if no such clustering would occur (Brown, Engberg and Matthews, 1967). Goodwin et al. call this phenomenon carrier-dependence and discuss its effect on their measurements of sensitivity and phase (1975).
The variance of the sinusoidal fit is comparable to figures published by Hulliger et al. (1977) for low frequencies. For higher frequencies (>50 Hz) the fit is not so good due to irregular filling of bins.
The responses of the model during the sensitivity analysis with sinusoidal stretches are comparable for dynamic and passive conditions but are very different during the ramp and hold stretches.This is due to the fact that under passive conditions the muscular part of bag_1 is stiff as a result of the occurrence of intrafusal stiction. Thereby, its mechanical effect lets itself compare to that of the fiber during dynamic fusimotor stimulation, generating high viscosity.

Comparison of the current spindle model with previous ones.
So far several muscle spindle models have been formulated (Angers, 1965; Chen and Poppele, 1978; Hasan, 1983; Hasan and Houk, 1975; Rudjord, 1970; Windhorst, Schmidt and Meyer-Lohmann, 1976). Most of these models assume sensitivity to first and second order derivatives of length, i.e. velocity and acceleration, in order to obtain results which resemble the characteristic spindle response to ramp-and-hold stretches. The initial burst is usually explained as the acceleration component, the dynamic response as the velocity component and the static response as a reflection of actual length.
The assumption that a great deal of the spindle physiology should be explained as due to the specific properties of the transducer process is challenged by recent physiological studies (e.g. Brown, Engberg and Matthews, 1967; Hasan and Houk, 1975a/b; Hill, 1968; Proske and Stuart, 1985; Poppele, 1985). Instead, more and more the view is established that most of the spindle physiology can be explained as being due to a mechanical interaction between the muscular and sensory parts of intrafusal fibers (Matthews, 1981).
Muscle spindle models making use of complicated sensitivities of the transducer-process (to first and second order derivatives) are, therefore, rightly criticized by Hasan and Houk (1975a/b). Their work led to the formulation of the most recent model, namely that of Hasan (1983). This model is constructed out of three different components. Firstly, out of a sensory

region which behaves as a simple spring. Secondly, out of a polar region which gives rise to dry friction as the main source of spindle non-linearity. And thirdly, out of a muscular component which obeys the non-linear force-velocity curve of Hill (1938). The sensitivity to velocity is assumed to be low. The model has five free parameters which have been chosen by trial and error.

The model of Hasan provides firing rates which closely resemble observed experimental data. Nevertheless, it has some disadvantages. One of its major drawbacks is that it is constructed too rigid to allow changes of fusimotor activity. The model can vary its sensitivity like spindles do (shifting from more dynamic to more static) but only by chosing a completely new set of values for the five parameters. So changes in fusimotor activity influence the properties of both the sensory region as well as of the intrafusal fiber.

Also, by testing the model of Hasan in our laboratory it became clear that the model is not flexible enough to provide reasonable firing rates for variable velocities of ramp-and-hold stretching. Within the range commonly used in physiological experiments, the model, for a fixed set of parameter-values, rapidly deteriorates (producing infinite firing rates) when the stretch velocity is increased, especially with respect to the amplitude of the initial burst.

Possibilities and limitations of the current spindle model.

There are two aims of the current muscle spindle model. In the first place, the spindle model is well calibrated for ramp-and-hold stretches, and provides experimental physiologists with the option to make an estimate of the muscle spindle response under experimental conditions for which these responses cannot be measured directly. In comparison with the model of Hasan (1983) the currently presented model has the advantage that it is fully defined for a wide range of stretching velocities, stretching amplitudes and fusimotor conditions.

The other aim of the muscle spindle model is to give insight into the internal physiology of the muscle spindle itself. We wished to investigate how the various hypotheses about spindle physiology may interact, combine and finally result in the Ia-afferent discharge. Therefore, the muscle spindle model presented in this paper has been constructed as a so-called structural model. This means that the model attempts to predict the function of the whole out of the known properties of its individual parts. In this case, primary afferent activity is explained as a product of observed mechanical behaviour of two different types of intrafusal fiber. By simulating intrafusal events and taking into account observations and hypotheses available in literature, it

has become possible to evaluate the relative importance of the hypothetical mechanisms to the functioning of the whole.

Indeed, as has been demonstrated by this study, a great deal of the characteristics of spindle response (viz. dynamic sensitivity, phase advance on sinusoidal stretches and the effects of fusimotor stimulation) can be explained within the scope of a mechanical model. Nevertheless, in the course of development of the model it became clear that some of the characteristic spindle response remains unexplained in terms of mechanical behaviour and it is shown that these properties need to be searched in the specific behaviour of the transducer and encoding-process.

By emphasizing not only the possibilities and achievements of the current model but also discussing its limitations, we hoped to contribute to the understanding of the complex relation between intrafusal mechanics and muscle spindle sensitivity. Emphasizing weaknesses of the current model may add in such understanding by three different ways.

Discrepancies between model and experimental results may give clear indications were the model fails and what improvements need to be made. For instance, the failure of the model to provide sufficient phase advance during sinusoidal stretching within the linear range of the passive spindle, indicates that the formulation of cross-bridge fixation within the spindle model is too stiff (allowing no change of length at all).

The process of modeling itself, may point out areas in muscle spindle experimental physiology which still provide insufficient information.

This last point brings us to what, in our eyes, are the main questions still to be solved: (1) What exactly is the relation between (a) the opening of the spiral endings, (b) the generator potential and (c) the firing rate, (2) where exactly does the encoder process take place, how does it work and why does it depend on the rate of change of the generator potential and (3) what happens during sinusoidal stretching within the linear range? Furthermore, for a proper understanding of the role of muscle spindles in gross motor function, it may be of interest to have a muscle spindle model in operation, simulating the average sensitivity of a whole group of muscle spindles rather than of the one coincidental exponent of this group that can be found in literature. Therefore, we are developing spindle models in close cooperation with experimental physiologists[11].

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[11] An example of such a project may become an investigation to assess the extent of a contribution of so-called stretch activation to the dynamic muscle spindle response (Boyd, 1976; Poppele and Quick, 1981; Dickson et al., 1989).

We believe that knowledge about muscle spindle physiology can only be advanced by translating and joining existing theories from various sources into one mathematical simulation. Uncertainties in the development of the model will shamelessly reveal gaps in our knowledge about spindle physiology. This leads, from sheer necessity, to the formulation of new questions to experimental physiologists. Ideally, therefore, the modeling and the development of new experiments go hand in hand.

k_1	Active Component Bag_1	$8.53 \cdot 10^{-2}$	FU*
k_2	Passive Component Bag_1	2.75	FU
k_3	Active Component Bag_2	$2.67 \cdot 10^{-1}$	FU
k_4	Passive Component Bag_2	4.81	FU
h	Sensitivity of Firing Rate to Change in Receptor Potential	$12.84 \cdot 10^{-2}$	pps/(mV/s)
e_1	Maximal dynamic Fiber Force Bag_1 (relative to isometric)	4.50	
e_2	Maximal dynamic Fiber Force Bag_2 (relative to isometric)	1.42	
b_1	Passive Fiber Damping Coefficient of Bag_1	$9.91 \cdot 10^{-3}$	FU/(mm/s)
b_2	Passive Fiber Damping Coefficient of Bag_2	$5.33 \cdot 10^{-3}$	FU/(mm/s)
f_e	Stretch Force Enhancement (relative to isometric)	$1.49 \cdot 10^{-1}$	

* FU = Force Unit, which is the force generated by the sensory zone when stretched 1 mm.

Table 2.1.:

Values for the model parameters which were obtained by computer optimization in order to let the model simulate experimental results of Crowe and Matthews (1964) as well as possible. Values in force units (FUs) are arbitrary since they can be scaled by a constant without change in behaviour of the model.

2.5. APPENDIX

A flow diagram illustrating the relation of the different Appendices in the model as a whole is given by figure 2.6.

A. Calculation of new nodal positions y_1 and y_2

In order to calculate primary afferent firing rates (Appendix C) it was necessary to know the lengths of both sensory zones, (y_3-y_1) and (y_3-y_2). As the procedure is similar for both nuclear bags the solution will only be given for the dynamic bag_1.

For the bag_1 component the length of the sensory region can be found by determination of the nodal position y_1. Nodal position y_3 is given by the experiment, since sinusoidal or ramp and hold stretches are externally enforced. Nodal position y_1 depends on the instantaneous dynamic force equilibrium at the node, which depends on the pulling force of the elastic sensory zone, counterbalanced by the force generated by the muscular part, which depends on its length, velocity and stimulation rate. The latter force was calculated using the muscle model as suggested by Otten (1987), with stretch force enhancement as an extra property. This model contains active force-length, passive force-length and force-velocity relations, that can be tuned depending on muscle architecture and fiber type. Since a single muscle fiber can be considered as a very small and simple muscle, the model can be used for intrafusal fibers.

In the spindle model the values for a number of parameters were obtained by computer optimization. These values were tuned in order to obtain a model's simulation to ramp-and-hold stretches which fitted data on real spindles as well as possible. The values of these parameters are listed in Table 2.1. The values for the remaining parameters were all derived from literature. These parameters have been listed in Table 2.2. together with their values and references.

Part of the fixed parameters, those defined by Otten (1987), were chosen to simulate extrafusal muscle fiber characteristics, such as the skewness of the active force-length curve, the roundedness of its top and its width (with values of respectively b=0.5, a=2.2 and s=0.2) and the shape of the passive length-force curve (c1=-10, c2=7, Fc=0.3). Optimal fiber length was set at 11.5 mm for both bags, so that all events occurred below optimal length.

The force equilibrium between muscular and sensory part generates a nonlinear differential equation, which is numerically solved using the Runge-Kutta method with adapted stepsize (Press, Flannery, Teukolsky and Vetterling, 1987).

For ramp-and-hold stretches the full time of testing is chosen three times the duration of the ramp. The total number of time increments is 128 and, therefore, ∂t is chosen three times the

ramp duration divided by 128. For sinusoidal stretches the total time for testing is three periods of the sine. Consequently, it is determined by taking 3 divided by the product of 128 and the frequency of the sine. If needed the adapted stepsize method divides individual time segments into smaller steps (Press, Flannery, Teukolsky and Vetterling, 1987).

parameter name:	parameter value:	source:
ltp[1]	5 mV/mm	[4] , [6] and [26]
ltp[2]	2.5 mV/mm	[4] , [6] and [26]
ptr	120 pps/mV	[4] , [6] and [26]
$V_{max}[1]$	5.9 lengths/s	[10] (slow twitch)
$V_{max}[2]$	14.0 lengths/s	[10] (fast twitch)
F_x	0.1	plots of modulation vs. amplitude (see text)
a	2.2	fitting active part of force-length relationship of extrafusal muscle fiber [35]
b	0.5	
s	0.2	
c1	-10	fitting passive part of force-length relationship of extrafusal muscle fiber [35]
c2	7	
fc	0.3	

Table 2.2.:

Values for the fixed constants of the muscle spindle model. In the last column references have been given to experimental data from which these values have been extracted (for explanation see text).

B. Correction for stiction

For the introduction of stiction, the length of the free section of the muscular part of the dynamic bag_1-fiber model needed to be updated several times during one time-increment. Depending on the velocity and amplitude of stretching a variable number of cross-bridge regions may yield during one timestep ∂t.

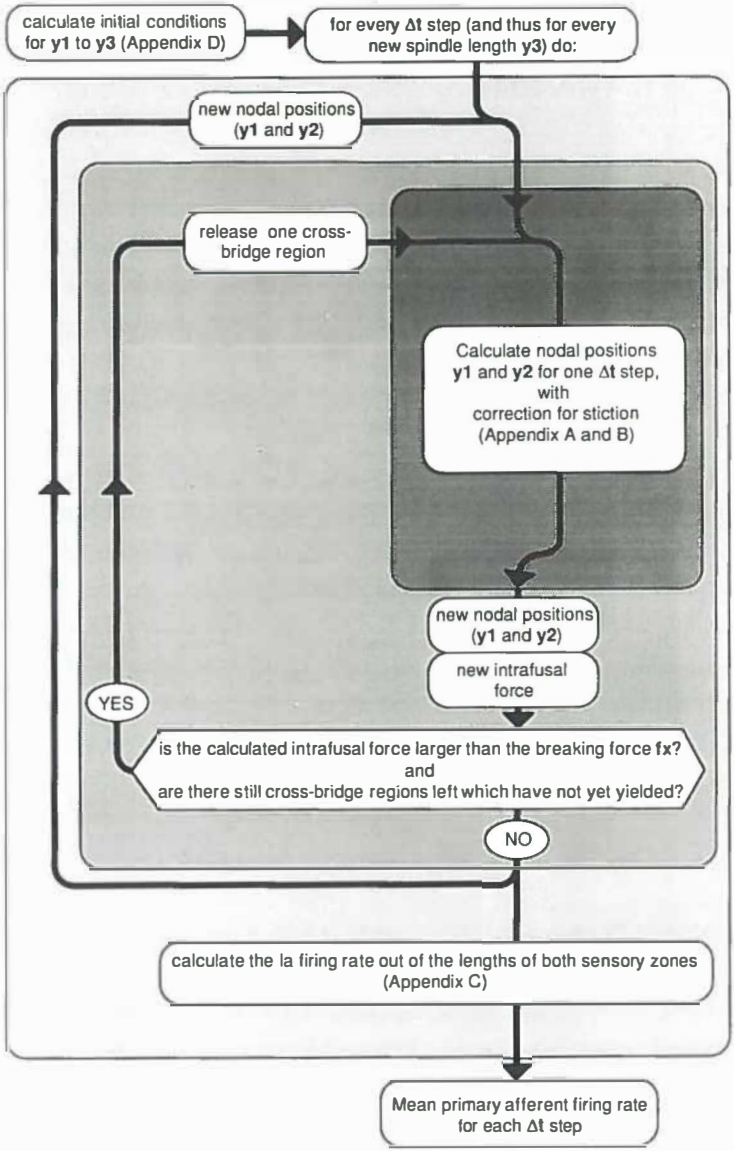


Figure 2.6.:

Flow chart of the computer programme which displays the relation between the different algorithms of the muscle spindle model. The exact mathematical formulations of the different algorithms are given by the respective Appendices.

Initially, all cross-bridge regions in the model were fixed. The muscular part had an initial length y_{xbr}, which was equal to the relaxation position y_1 of the muscle spindle at its starting-length (Appendix D). On stretching, the calculated intrafusal force gradually increased until it exceeded the breaking force F_x. At this stage one of the one hundred cross-bridge regions yielded. Several changes were then taken into account.

Firstly, the point of attachment of the muscular part (y_x), from its starting position y_{xbr}, moved to the left (Fig. 2.1a.) one percent of y_{xbr}. Secondly, the instantaneous free length and optimal length of the fiber were recalculated. These values were used to recalculate the intrafusal tension (3a) with the muscle fiber model and it was checked whether this tension exceeded F_x. If so, yet another cross-bridge region yielded. If not, a new state of equilibrium was reached, and the firing rate of the primary afferent was determined out of the calculated length of the sensory region of the bag_1 and that of the bag_2 (equation 1 and 2).

By gradually breaking down all cross-bridge regions the model of the dynamic bag_1 fiber returns to its simple version without stiction. This implies that, if F_x is chosen suitably, only the sensitivity of the model for small stretches will be increased whereas the sensitivity for large ramp-and-hold stretches will remain almost unaffected.

By experimenting with different values for the breaking force F_x it appeared that, in modulation versus amplitude plots (see Fig. 2.4a.), the value for this parameter not only determined the upper boundary of the linear range (the amplitude of stretching at which the first group of cross-bridges yielded) but also influenced the level at which the modulation settled for amplitudes beyond this linear range. The value of F_x was chosen to obtain the best model's results with respect to three criteria: the upper boundary of the linear range, the cross-over amplitude and the modulation depth for sinusoidal stretching outside the linear range.

C. Calculation of Ia firing rate out of the lengths of both sensory zones

Primary firing rates (R_{Ia}) were calculated out of the lengths of the sensory regions of the bag_1 and bag_2-fibers, by a formula simulating competition between both nuclear bags (see Fig. 2.1.). An intermediate step is to calculate the generator potentials P_{Ia1} and P_{Ia2}:

(1) $P_{Ia1} = ltp_1 . ((y_3-y_1) - zl_1) ; \quad P_{Ia2} = ltp_2 . ((y_3-y_2) - zl_2)$

This formula provides a very sharp competition between both bags.

The primary firing rate can then be calculated with

(2) $R_{Ia1} = ptr * P_{Ia1} + h * d P_{Ia1}/dt$; $R_{Ia2} = ptr * P_{Ia2} + h * d P_{Ia2}/dt$

(3) if $R_{Ia1} > R_{Ia2}$ then $R_{Ia} = R_{Ia1}$ else $R_{Ia} = R_{Ia2}$

The two constants, zl_1 and zl_2, are so-called zero lengths of the sensory regions. These zero-lengths denote the lengths of the respective sensory zones below which the contribution to the generator potential is zero.

Both zerolengths have been calculated in order to obtain a firing rate of exactly 0 pps when the spindle is kept at a length of 10 mm under passive circumstances (γ_d and γ_s are 0) and were calculated with a golden section search procedure (Press, Flannery, Teukolsky and Vetterling, 1987), with force equilibrium as objective, so that at the onset of a simulated experiment, the nodal positions would not creep from their start positions.

D. Calculation of the initial positions y_1 and y_2 .

At the onset of a simulation, the model was initialized with starting values for y_1 and for y_2 given by a golden section search procedure (Press, Flannerly, Teukolsky and Vetterling, 1987) looking for a force equilibrium at the given starting length and fusimotor condition

As indicated in Appendix B the values y_x and y_{xbr} were both set equal to the initial value of y_1. In other words, crossbridge regions of the muscular part of the dynamic bag_1 fiber were assumed to have become fixed prior to stretching.

CHAPTER 3

Mechanical impedance, stiffness and visco-elasticity

3.1. INTRODUCTION

Altogether, the elbow joint is controlled by five different muscles, which, as the elbow is a cylindrical joint, can be subdivided into two functional groups: the elbow extensors (mm. triceps, anconeus) and the elbow flexors (mm. biceps, brachialis and brachio-radialis). By controlling the level of activation of both muscle groups, the central nervous system (CNS) regulates the joint angle and the joint stiffness; joint angle is controlled by the difference in activation of flexor and extensor muscles (Bizzi, Accornero, Chapple and Hogan, 1982a/b); joint stiffness by the parity in their activation (Humphrey and Reed, 1983).
In other words, any angle of the human elbow joint is maintained by a balanced tuning of antagonistic muscle activity. At a fixed elbow angle, the force exerted by both muscle groups depends upon a muscle's length as well as its activation. Bizzi et al. indicate that for any pair of agonist-antagonist muscle activations the joint angle is determined by the position for which the antagonistic muscle torques are in equilibrium; or, at which an intersection occurs of their torque-angle relationships.
According to the diagrams of Bizzi et al., one pair of antagonistic muscle activations generates a single equilibrium position. The reverse, however, is not true: one particular joint angle may be maintained by many different combinations of antagonistic muscle activation. If the joint is in equilibrium for one pair of antagonistic torques it will also be in equilibrium for any multiple of this pair. Consequently, one joint angle can be maintained at different levels of co-contraction. The level of antagonistic co-contraction determines the stiffness of the joint (Humphrey and Reed, 1983).

The present and following paper investigate, by imposing perturbations on the human forearm, the way by which angle and stiffness of the elbow joint are controlled. There is an important

distinction between the (mechanical) effects of the pre-setting of antagonistic muscle activation, prior to an imposed perturbation, and of changes in antagonistic muscle activation, occurring in response to an imposed perturbation. Therefore, it was decided to describe the experimental findings in two papers separately: the first paper deals with data on joint stiffness determined by the pre-setting of antagonistic muscle activation; the second paper deals with data on the control of elbow joint angle after a perturbation, as changing joint angles depend on changes in muscle activation.

Elbow joint stiffness (or joint impedance or joint visco-elasticity[1]), which is the subject of the present paper, is commonly investigated by measuring the resistance when perturbing the arm. This parameter is studied, either while the elbow muscles are electrically silent (e.g. Boon, Hof and Wallinga-de Jonge, 1973; MacKay, Crammond, Kwan and Murphy, 1986), or when they are actively contracting against a pre-load (e.g. Colebatch and McCloskey, 1987).
In the present study, we demonstrate the effects of differences in the level of antagonistic co-contraction upon the stiffness of the human elbow joint. We were able to vary the level of antagonistic co-contraction by either withholding or providing support to a subject's forearm. Without arm support, subjects displayed a moderate level of co-contraction in the electromyograms (EMGs) of elbow antagonists; whereas, with arm support, little or no electrical activity was observed. Under both conditions, subjects were instructed not to respond to perturbations imposed, so that they did not deliberately increase the stiffness of their elbow joint.

3.2. MATERIALS AND METHODS

Data were collected by imposing perturbations on the forearms of ten healthy human volunteers (aged 26 to 52, two females and eight males). All subjects gave their informed consent and, except for one of the authors, were unaware of the hypotheses tested.

[1] The mechanical joint impedance is used to indicate the net resistance encountered when perturbing the forearm, which arises from (1) the forearm moment of inertia, from (2) joint viscosity and from (3) forces exerted by the (relaxed or contracting) muscles crossing the joint. Joint visco-elasticity designates the limb resistance arising from (2) and (3), thus when the effects of the limb's moment of inertia are excluded. Finally, joint stiffness (or elasticity) is the resistance of the limb against a perturbation which is only due to the limb's elastic properties and, consequently, does not depend upon the perturbation velocity.

The perturbation apparatus

The subjects sat in a dental chair their left arm fixed in a perturbation apparatus as shown in figure 3.1a.

The wrist was fixed by means of adjustable plastic clamps to an armature (Fig. 3.1a.: 2) which allowed movement in a horizontal and vertical plane. This was achieved by mounting the

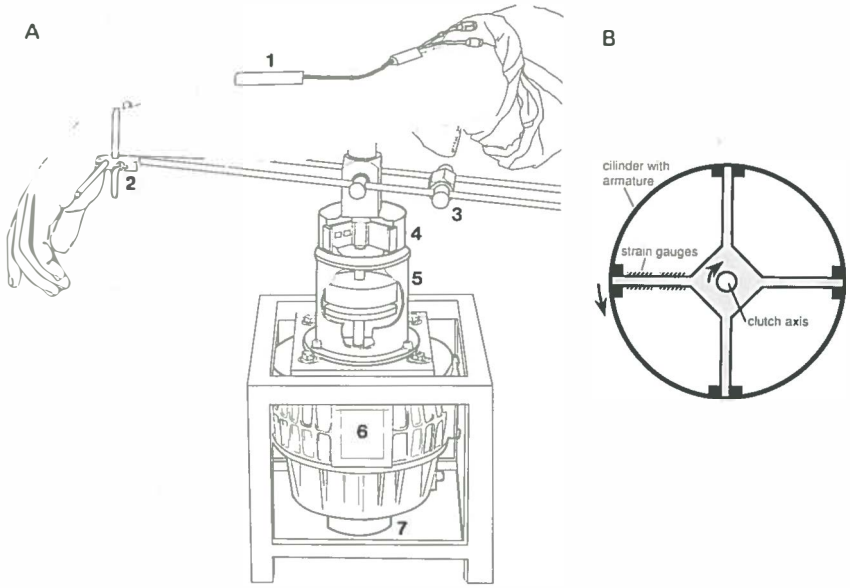


Figure 3.1.:

A: the experimental apparatus used for studying posture maintenance at the human elbow: 1. goniometer signalling the elbow joint angle; 2. armature; 3. counter weight correcting for the weight of the armature but not for that of the arm; 4. torque sensor; 5. clutch; 6. torque motor; 7. potentiometer signalling the motor position; B: schematic representation of the torque sensor; this sensor was composed of four arms connected to the clutch axis and falling into slots on the inside of a cylinder. Two pairs of strain gauges were glued to either side of one of the arms. See text for a further description.

armature hinged to a metal block driven by a torque motor displayed at the bottom part of figure 3.1a. (6). On top of the metal block was a plastic cup, providing support to the medial epicondyle of the subject's arm, so that the rotatory axis of the elbow joint was positioned in the axis of the motor. Prior to each experiment the armature was adjusted in length so that the hand

could flex around the wrist, hanging in pronation over the end of the armature. After adjusting the length of the armature, the arm was removed from the apparatus and a contra-weight (Fig. 3.1a.: 3) was positioned so as to balance the weight of the armature. In this way we corrected for the mass of the armature but not for that of the arm.

The position of the motor-axis of the torque motor (signalled by a potentiometer underneath (Fig. 3.1a.: 7)) was at all times controlled by an analogue servo-mechanism of which the set-point was derived from a computer system. To obtain a constant velocity movement of the motor, the computer generated an array of positions (with equal spacing) and converted this array into an analogue signal which was fed into the position servo. The servo-system usually displayed some oscillation in its position regulation, visible in the recording of the torque. These oscillations counteracted the phase lead or phase lag of the trajectory of the motor (and arm) with respect to the trajectory aimed at.

The axis of the motor was fixed to the axis of an electrical clutch (Fig.3.1a.: 5), which allowed the motor to be coupled or uncoupled from the subject's arm. If the clutch was turned off, the arm could move freely, experiencing minimal resistance from the moment of inertia of the armature or from dry friction of the rotatory axes. The condition of the clutch (on or off) was also controlled by the computer system.

The force transmission through the clutch was limited by the maximal gain of the servo system controlling the motor position. Torques achieved were maximally 2.6 Nm for 'flexion' and 3.3 Nm for 'extension'. This maximal force transmission sufficed to give forceful jerks to the arm. Any further increase in gain of the position servo was decided undesirable for safety reasons. In some experiments, a Y-shaped support was placed under the armature so that the forearm could rest upon the apparatus.

Our subjects were asked to place the medial epicondyle of the left arm on the cup on top of the metal block mentioned earlier (Fig.3.1a.). They were instructed to keep their arm with 90 degrees abduction at the shoulder and 90 degrees flexion at the elbow joint. In the cases when the armature was not supported, subjects had to hold their forearm actively in a horizontal plane, opposing gravity. This introduced electrical activity in the shoulder muscles: the posterior part of the m. deltoideus and the mm. infraspinatus and teres minor contracted to counteract the endorotating torque at the shoulder caused by the weight of the forearm. This was also accompanied by a moderate activity in the mm. biceps, triceps and brachio-radialis, although contraction of these muscles exerts little or no exorotating torque at the shoulder joint.

One can argue that, by the experimental arrangement, the tilting forearm also exerts a torque at

the elbow joint which threatens to turn the proximal end of the radius away from its articular surface on the humerus. This (sub-)luxation, however, is prevented by the capsule of the joint (Gray, 1980) and not by a contraction of the elbow muscles which are more concerned with the control of forearm position than maintaining the integrity of the elbow joint.

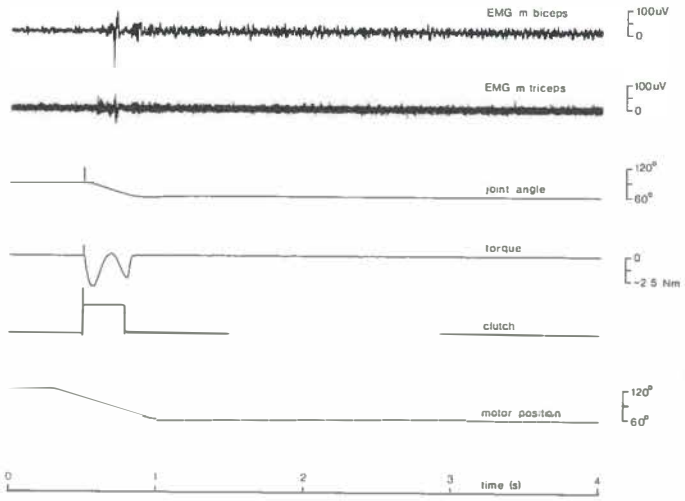


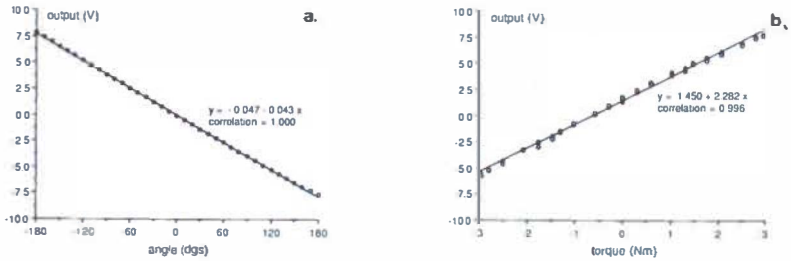
Figure 3.2.:

Full data recording of an arm perturbation (subject MB). Whilst the elbow joint is maintained at an angle of ninety degrees flexion, the motor starts moving. When the motor has reached the velocity aimed at, the clutch is suddenly turned on and, somewhat later, released again. Only when the clutch is turned on can the motor drive the arm. When the clutch is turned off, the arm experiences minimal resistance from the perturbation apparatus. In this particular case, the arm stabilized quite rapidly at a position shifted with respect to the starting position. The top two traces display EMG recordings from the mm. biceps and triceps. Note the increase in tonic EMG activity of the m. biceps.

Consequently, not allowing a subject's arm to rest on the apparatus imposed a physiological pre-load on the arm, which was, in general, accompanied by a co-contraction of elbow antagonists, although this load could only minimally be opposed by these muscles.

The subjects were asked to sit as relaxed as possible in the position described above and not to

respond to the movements imposed on the forearm. We applied fifty perturbations to each subject's forearm. These perturbations were randomized with respect to their velocity (ranging from ±20 dgs/s to ±150 dgs/s) and to their amplitude (ranging from -30 to 30 degrees).
In two of our subjects, we studied the mechanical joint impedance in more detail, by using a constant perturbation velocity (±75 dgs/s). The outcome of these experiments was compared with similar experiments performed when the arm, in contrast, was supported; with arm support, the subjects' elbow muscles silenced completely.
There are several reasons why we are confident that the subjects' responses were automatic and involuntary. Firstly, subjects could not anticipate a perturbation since its onset and direction was randomized. Secondly, during the experiments, we started vivid conversations with the



goniometer output torque transducer output

Figure 3.3:

a. Calibration plot illustrating that the output voltage derived from the Penny & Giles goniometer was a linear function of its bending angle.
b. Calibration plot illustrating that the output voltage of the torque sensor was a linear function of the imposed torque; torques were tested over a range of -3 to 3 Nm, which roughly equalled the minimal and maximal torques attained by the motor.

subjects and found no changes in behaviour of the arm, irrespective of whether the forearm was supported or not. Finally, subjects expressed their surprise that they were unable to tell whether the arm had been moved either by the motor or by themselves. That subjects, when appropriately instructed, can indeed refrain from voluntary action on the limb under study, is also confirmed by Crago et al. (1976).
The way by which a perturbation was carried out is illustrated by figure 3.2. Before each perturbation, the motor brought the arm to a starting position of 90 degrees elbow flexion, then

the clutch was turned off. Usually, the subjects spontaneously maintained the acquired arm position. At the start of each perturbation, the motor was put into motion with a velocity determined by the computer programme, and, after a delay of roughly 0.2 s, the clutch was activated for a particular period of time (in the range of 0.01 to 1.5 s).
The sudden transmission of motor torque through the clutch caused an abrupt perturbation of the forearm. After the clutch had fully detached, the arm could move freely, and, usually, its trajectory displayed a damped oscillation.

Elbow angle measurement
The angle of the elbow joint was monitored by means of a goniometer (M180, Penny & Giles Blackwood Ltd, UK) fixed to the subject's arm by two sided adhesive tape (Fig. 3.1a.: 1). After amplification by a bridge-amplifier, this goniometer provided an output voltage which was a linear function of its flexing angle (Fig. 3.3a.). Its precision was better than ± 0.5 deg.

Torque measurement
Between the clutch and the armature was a torque transducer (Fig. 3.1a.: 4). When the axis of the clutch was enforced, the motor torque was transmitted through the clutch and through the torque transducer, to the subject's arm on top. The torque exerted was sensed by four balanced strain gauges glued in pairs to either side of one of a set of four arms attached to the axis of the clutch (Fig. 3.1b.). These arms fitted into four slots on the inside of a cylinder (open at its bottom end). Force transmission through the torque transducer resulted in minimal bending of its four arms, which was picked up by the strain gauges and fed into a bridge amplifier. The output voltage derived from the bridge amplifier was a linear function of the imposed torque (Fig. 3.3b.).
By its construction, the calibration of the torque sensor remained independent of the length of the armature (which was adapted to the size of the subject's forearm). The torques recorded could be directly related to the signal from the goniometer as we recorded the joint angle of the elbow directly from the subject's arm itself.

EMG recording
There are five muscles controlling the angle of the elbow joint. In the present study we have constrained our EMG analysis to the principle agonist and antagonist, relying on surface recordings of the mm. biceps and triceps brachii. The m. anconeus was not studied as its

contribution to elbow extension was assumed to be small, due to its limited physiological cross-sectional area (less than 20% of the m. triceps; An, Hui, Morrey, Linscheid and Chao, 1981). Neither did we investigate the m. brachio-radialis since recordings of this muscle showed that its EMG activity varied only little as function of elbow angle (during pilot experiments at our laboratory). Finally, the m. brachialis was excluded from further study as its EMG activity is reported roughly comparable to that of the m. biceps (Gray, 1980).

Surface EMGs of m. biceps and m. triceps were measured either from two neonatal (Consolidated Medical Equipment Inc., New York USA) or from two pediatric (HP 40426A, Hewlett Packard USA) disc electrodes fixed, with a spacing of approximately 5 cm, over the bellies of either muscle. By means of cross-correlation analysis it was ensured that no major cross-talk occurred between both recordings.

Signals derived from the electrodes were amplified by a laboratory designed EMG amplifier (1000 x amplification, optically isolated, optional DC or AC, optional 50 Hz filter with -3 dB points at 32 and 72 Hz, noise < 10 μV pre-amplification, bandwidth DC: 0 Hz to 4.3 kHz and AC: 10 Hz - 4.3 kHz).

To check the tonic EMG activities during an experiment we recorded, for five of the ten subjects, the entire experiment on tape. Apart from EMG responses to the imposed perturbations and to the re-positioning of the arm in between, we did not notice any marked changes in the basic level of EMG-activity (for instance, due to fatigue).

Data registration and processing

The following data were obtained from the subject and the perturbation device (Fig. 3.2.):

a. the position of the motor-axis.

b. the condition of the clutch,

c. the torque exerted by the motor on the subjects arm (only when clutch was activated),

d. the joint angle of the subject,

e. the EMG activity recorded from the subject's biceps and triceps muscles.

All data were digitized and stored on the hard disc of an Apple counterwe II computer, equipped with a NB-DMA-8 and a NB-MIO-16L board. Experimental set-up as well as data recording and processing ran on software developed at our laboratory within a LabView environment (release 2.0, National Instruments, Texas USA). Data were sampled with a frequency of 1 kHz and directly stored on hard disc. The sampling frequency was 9.6 kHz when EMGs were recorded.

Calculation of the mechanical impedance, the stiffness and the visco-elasticity of the elbow joint

Although we disposed of the torques required to move the arm over a certain angle, we were unable to calculate the stiffness (S) of the elbow joint out of the ratio $dT/d\varphi$ (T denotes the torque and φ the joint angle), since the torques generated by the motor were of a limited range. (Torques larger than 2.6 Nm for flexion and 3.3 Nm for extension could not be generated, while Ma and Zahalak (1985) show that, for the actively contracting m. triceps, step changes in elbow angle produce changes in torque which easily reach values of 10-20 Nm.)

Therefore, we decided to calculate joint stiffness indirectly, by calculating the energy required to perturb the forearm. For this purpose, we calculated the total amount of energy the motor put into the forearm. To eliminate all possible reflex actions we calculated joint stiffness only during the first 100 ms after perturbation onset (this will be explained in the results section). Over this first 100 ms of perturbation the so-called input energy (E_m) amounted:

$$(1) \qquad E_m = \int T \, d\varphi$$

Since part of this input energy was used to accelerate the arm and armature, the input energy calculated depended upon the mechanical joint impedance. We, therefore, subtracted the effects of arm acceleration.

In order to calculate the kinetic energy of the moving parts we, firstly, calculated the moments of inertia of the arm and armature. For that, we subdivided each subject's arm into 10 sections of which the volume was determined by a gradual submersion of the arm in a known volume of water. The volume of each section was multiplied by the (average) specific mass of the arm (1.115 kg/dm^3; Gowitzke and Milner, 1988) and the moment of inertia of the arm (I) was calculated according to:

$$(2) \qquad I = \sum_i M_i \cdot r_i^2$$

(where M_i denotes the mass of a section i and r_i the distance from its centre of gravity to the centre of rotation).

The estimate of the moment of inertia is a crude one due to the large contribution of the hand of which the mass could be measured with reasonable accuracy, but of which the distance from the centre of gravity to the joint axis could only be determined by approximation. Nevertheless, the calculated moments of inertia were within the range of those calculated by others (e.g. Ma and

Zahalak, 1981)[2].

The moment of inertia of the arm was further increased by the moment of inertia of the counterweight (mass: 0.3 kg) and that of the armature (mass: 0.1 kg). These were also calculated by applying equation 2. Armature and counterweight increased the moment of inertia of the arm by 10 to 15 percent. The contribution of the torque transducer to the total moment of inertia was ignored as its distance to the rotation-axis was small. The values for the calculated moments of inertia for each subject are provided by Table 3.1.

Making use of the calculated moments of inertia of arm and armature we calculated the kinetic energy (E_k) of the moving parts 100 ms after onset of the perturbation:

$$(3) \qquad E_k = {}^1/_2 \, I \, (\varphi')^2$$

with φ' the velocity of the arm (rad/s) calculated by taking the first order differential from the position signal.

As expected, not all the input energy was turned into kinetic energy, a considerable proportion was absorbed by the muscles crossing the elbow joint. The energy absorbed by the muscles (E_δ) was calculated by subtracting the kinetic energy from the total input energy:

$$(4) \qquad E_\delta = E_m - E_k$$

This energy (δ energy) is absorbed due to muscle elasticity and muscle viscosity. Both factors are known to increase with increasing muscle activation (Hill, 1938; Aubert, 1956; Otten, 1987). Therefore, the δ energy contains information about the level of co-contraction at the joint and thus of the joint stiffness.

Our data on δ energy as function of forearm rotation fitted a parabolic relation. This allowed us to calculate one characteristic figure of apparent joint stiffness for each subject, under the simplification that the forearm is a body held in position by two antagonistic linear springs. The

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[2] In one subject, we made an estimation of the uncertainty in our calculations of the forearm moment of inertia. For this subject, the volume of the hand was 0.35 dm^3 (mass = 0.39 kg) on a total forearm volume of 1.63 dm^3. It was assumed that the distance from the hand's centre of gravity to the elbow joint axis (roughly 30 cm) was estimated with an uncertainty of 1 cm. So, we calculated the contribution of the hand to the overall moment of inertia for a distance of 29 cm and of 31 cm to the elbow axis. In the first case the moment of inertia amounted 0.39 x $0.29^2 = 0.0328$ kg·m^2, in the second 0.39 x $0.31^2 = 0.0375$ kg·m^2. So, the uncertainty in the overall moment of inertia (0.0962 kg·m^2) amounted ± 0.0023 kg·m^2 (± 2.5 %).

Subject:	inertia arm:	inertia armature:	median joint stiffness:	10th percentile:	90th percentile:
	($kg \cdot m^2$)	($kg \cdot m^2$)	(Nm/rad)	(Nm/rad)	(Nm/rad)
FK	0.0427	0.0073	8.14	-16.58	15.09
MeB	0.0476	0.0069	11.40	-9.53	18.11
IN	0.0528	0.0065	7.58	0.61	12.23
JeB	0.0571	0.0080	14.36	9.95	22.79
MaB	0.0578	0.0089	10.55	4.05	27.95
RD	0.0658	0.0111	10.27	-47.22	15.09
AS	0.0825	0.0090	13.56	10.95	21.30
JaB	0.0857	0.0105	13.33	6.80	35.79
HN	0.0907	0.0106	14.67	5.00	38.42
SC	0.0913	0.0107	14.09	8.07	50.14
all	-	-	*11.84*	*3.62*	*25.77*

Table 1:

Overview of the data obtained from studies on ten subjects. Units are given between brackets. The meaning of the data is discussed in the text.

change in elastic energy (E_p) stored by these two springs (with spring constants k_1 and k_2; k_1 positive and k_2 negative) on a rotation φ was derived from the equation:

$$(5) \qquad E_p = \tfrac{1}{2} \cdot (k_1 - k_2) \cdot \varphi^2 \qquad (k_1 > 0, k_2 < 0)$$

The parameter $k = k_1 - k_2$ was calculated for each pair of φ and E_p and represented the apparent elbow joint stiffness. For each subject, we provided the median value as well as the 10th and 90th percentiles for calculated k's over the full set of perturbations (Table 3.1.).

Though the above mentioned method is a suitable way to compare data between our ten subjects, it remains incorrect: the calculated δ energy is not only dependent on arm rotation, it also depends on arm velocity. Therefore, the actual δ energy depends also upon joint visco-elasticity and not upon joint stiffness alone.

Another point of concern was the height of the shoulder joint with respect to the support of the

medial epicondyle. A change in position of the shoulder causes tilting of the elbow joint axis and the forearm to move in a plane deviating from the horizontal. In this situation, the centre of gravity of the forearm is displaced vertically and a rotation of the forearm may cause changes in its potential energy. Tilting the plane of movement by 10 degrees, causes a rise in potential energy of about 0.0015 J per 5 dgs arm rotation[3]. This is only slightly more than 1 percent of the δ energy measured at 5 dgs arm rotation. It was, therefore, concluded that small differences in the height of the shoulder would not cause a significant error in the calculated δ energy.

Data display

Data were retrieved from hard disc and, subsequently, visualized either by means of a laserwriter (Apple Macintosh II NTX) or by means of a x-y-plotter (DXY-980A, Roland DG Corp., Japan).

3.3. RESULTS

We tested our experimental set-up and the analytical techniques attached by imposing perturbations on a dummy load (a block of wood, of dimensions 0.368 m, 0.070 m and 0.054 m and with a mass of 0.607 kg) and comparing the outcome with the results obtained from perturbations of the human forearm. Figure 3.4. gives the results; data were plotted starting from clutch activation up to 100 ms after movement onset.

A comparison of dummy load and human arm shows that, for similar perturbation torques, the dummy is more rapidly accelerated than the arm. This is not surprising as the moment of inertia of the first ($I = 0.0358$ kg·m^2) was smaller than that of the latter ($I = 0.0857$ kg·m^2). Nevertheless, important differences are observed at a later stage of the perturbation: after the initial acceleration, the torque required to perturb the dummy rapidly turns to zero, whereas that required to move the arm does not or does but significantly later.

[3] Let the centre of gravity of the forearm move in a plane tilted 10 degrees with respect to the horizontal and let the centre of gravity reach its lowest vertical position when the arm is 90 degrees flexed at the elbow. If the distance from the forearm's centre of gravity lies at 0.15 m from the elbow joint axis, this would mean that the centre of gravity is displaced from the horizontal plane by $0.15 \times \sin(10^o) = 0.02605$ m, when the arm is rotated from maximal extension to 90 degrees arm flexion.
As the vertical movement of the centre of gravity changes with the cosine of the arm rotation, a rotation of 5 dgs would cause a vertical displacement of $(1 - \cos(5^o)) \times 0.02605$ m = 0.00010 m. For a forearm mass of 1.5 kg such a displacement represents a change in potential energy of 1.5 x 0.00010 x 9.8 (constant of gravity) = 0.0015 J.

The torque signal largely depends on the motor servo: the more the position of the motor axis deviates from the intended trajectory, the larger the corrective torque will be. Abruptly loading the motor with the mass of the dummy (or the arm), causes it to decelerate. The increasing error between intended and actual motor trajectory, causes a gain in torque until the dummy (or the

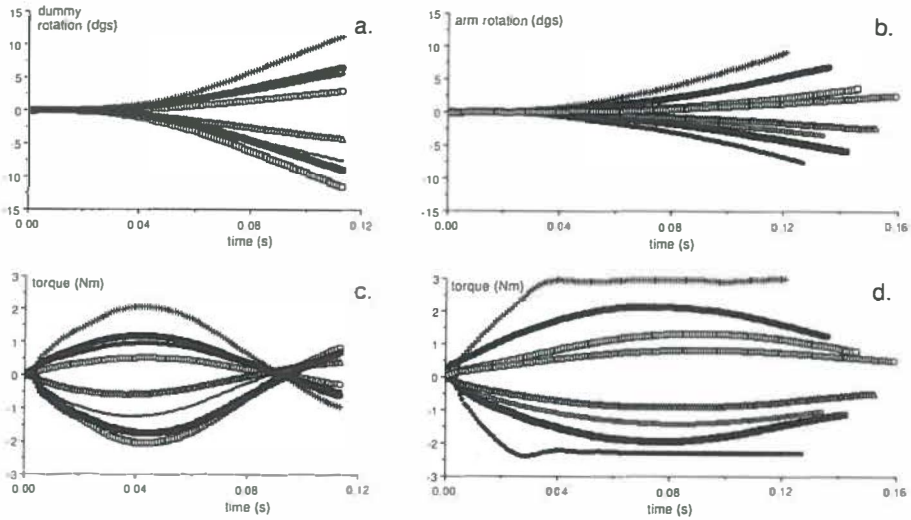

Figure 3.4.:

Plots for a series of perturbations imposed on a dummy load (a and c; I = 0.0358 kg·m^2) and on the arm of a human subject (b and d; subject: JaB; I = 0.0857 kg·m^2). For the dummy load and for the subject the same symbols were used to plot corresponding data on one perturbation. Data were obtained at 1 ms intervals and were all plotted in the graph. Only the first 100 ms of each perturbation is shown. For further explanation see text.

arm) is moved in concert with the motor. In the case of the dummy, the corrective torque of the motor was initially too large, and the motor started to move in advance of the trajectory aimed at. This caused the torque to become negative. In contrast, with the arm the motor servo was not capable of fully compensating for the increased load: the torque signal remained positive throughout the duration of the perturbation (not illustrated).

Figure 3.5. shows the results of our analysis of the mechanical impedances of dummy and arm. In contrast to figure 3.4., all graphs are now plotted as a function of dummy or arm rotation.

Negative torques are inverted for presentation reasons. To show the torque required to move dummy or arm over a particular rotational angle, we replotted for figures 3.5a. and 3.5b. the data of figure 3.4. A comparison of figures 3.5a. and 3.5b shows that for the arm more torque introduces less rotation. On first sight, these differences were not surprising since the arm had a greater moment of inertia than the dummy. However, whether this was the only explanation for the observed differences, required further investigation.

Figures 3.5c. and 3.5d. display the amount of energy per degree rotation put into the dummy or arm by the torque motor. These figures were obtained by the integration of figures 3.5a. and 3.5b. (see Materials and Methods); they display the surface below each torque-angle relationship. As can be seen by comparing figures 3.5c. and 3.5d. with the next four figures, part of the input energy was transformed to kinetic energy of the moving bodies, and part of it, at least in the case of the human arm, was absorbed by the muscles at either side of the joint.

Figures 3.5e. and 3.5f. display the amount of kinetic energy represented by the moving bodies. The calculation of the kinetic energy was based upon calculations on the moment of inertia of dummy and arm, together with data on 'arm' velocity, which were calculated from the position signal. Subtracting the kinetic energy from the total input energy provided a so-called δ energy (Figs. 3.5g. and 3.5h.).

For the dummy no significant δ energy was obtained, which was expected as there were no other components (except for minimal dry friction) to absorb the motor output energy. In contrast, for the arm a definite residue was found, indicating the absorption of energy by the visco-elasticity of muscles crossing the elbow joint. In this particular case, δ energy was relatively independent of perturbation velocity, as can be seen by the overlap of data on different trials despite differences in forearm velocity (compare the Figs. 3.5h. and 3.5f.).

The conclusion from this analysis of joint impedance was that the human arm, in contrast to a dummy load, was capable of absorbing energy other than kinetic energy by the visco-elasticity of the muscles crossing the joint. The method of analysis allows the quantitative determination of the relative contribution of (passive) mechanical and (active) muscular factors to the overall joint impedance.

Figure 3.6. shows the data on δ energy for a full series of perturbations imposed on the arm of one of our subjects. The data presented in this figure were gathered exactly 100 ms after the onset of arm movement; they correspond with the final points reached during the perturbations plotted in figure 3.5h. With some scatter, these data fit a parabolic curve also drawn in figure 3.6. The significance of this parabola is no more than to illustrate what would be the δ energy

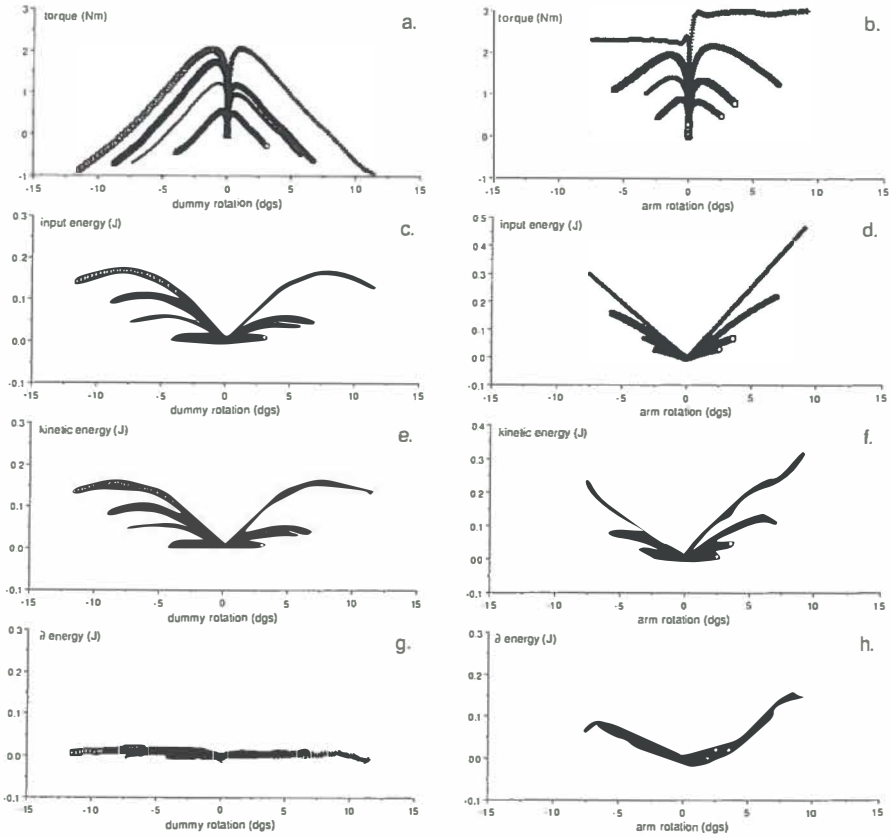

Figure 3.5.:

Processed data of the perturbations displayed in figure 3.4.. The data of the figures a,c,e and g were obtained from the dummy load, those of the figures b,d,f and h for the human subject. Again the same symbols were used to plot corresponding data (obtained at 1 ms intervals) on one perturbation. Plots of torque (a and b), input energy (c and d: $\int M \, d\varphi$), kinetic energy (e and f: $^1/_2 \, I \, \varphi'^2$) and δ energy (g and h) against angular displacement for different perturbations. Only the first 100 ms of each perturbation is shown. For further explanation see text.

expected from two opposing linear springs with a net stiffness of 13.3 Nm/rad (see Materials and Methods section).

Figure 3.6. suggests unjustly that the δ energy is only a function of arm rotation. It should, however, be emphasized that differences in the measured amount of arm rotation, also indicate differences in mean arm velocity. Thus, the gain in δ energy on increasing arm rotation may also be the result of an increase in arm velocity.

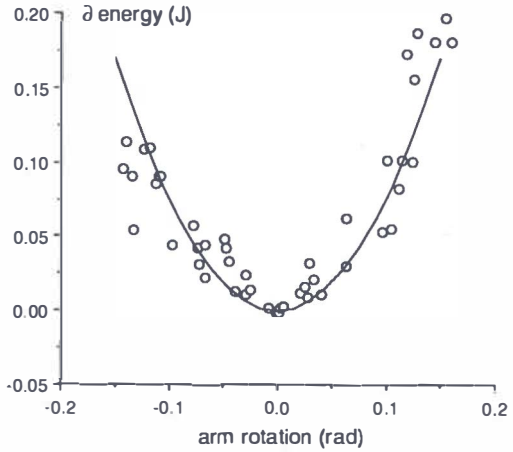

Figure 3.6.:

Plot of the energy absorbed by the antagonistic muscle groups as a function of arm rotation for a series of perturbations (subject: JaB). Only the final values of δ energy after the first 100 ms of each perturbation were plotted. A parabolic curve fitted through these data (continuous line) displayed the expected δ energy for the subject's median joint stiffness which amounted 13.3 Nm/rad.

To illustrate this point, we have plotted the data on δ energy in a three dimensional graph (Fig. 3.7.). This figure shows that differences in amplitude (reached 100 ms after perturbation onset) are accompanied by differences in perturbation velocity. The relation between these two variables is roughly parabolic (as for a simple mechanical load submitted to a constant acceleration). Also, this figure shows that the δ energy is not necessarily symmetrical for flexing and extending perturbations. For this particular subject the energy absorbed by the antagonistic muscles was larger when the m. biceps was stretched (and the triceps released) than the other way round.

The next point to ascertain was whether the interval of 100 ms was sufficiently short to prevent the contamination of δ energy by possible reflex activity. For this purpose, we selected two perturbations imposed on the arm of one subject and studied in detail the effect of changes in EMG activity on our calculations of δ energy. The figures 3.8a. and 3.8b. show two

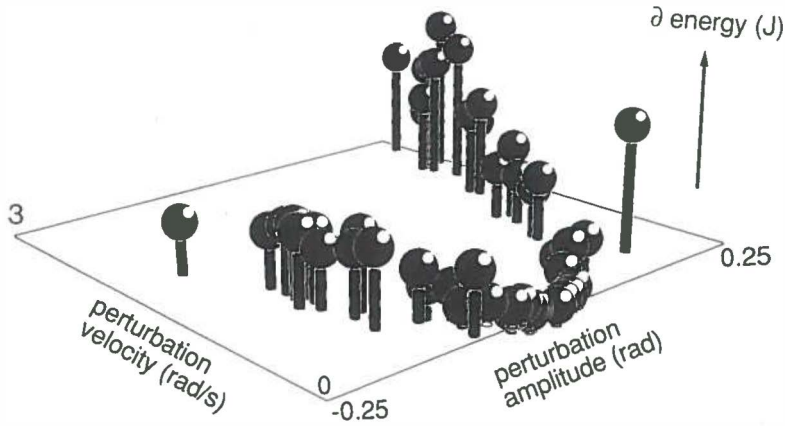

Figure 3.7.:

Three dimensional plot of the calculated δ energy (vertical axis) as a function of perturbation amplitude and perturbation velocity (Subject: SC). This plot shows that, because the δ energy was calculated at a fixed time interval 100 ms after perturbation onset, different perturbation amplitudes also indicate different perturbation velocities. The relation between perturbation amplitude and velocity is roughly a parabolic one (as expected for a simple mechanical load subjected to a constant acceleration). In this particular subject, the energy absorbed by the antagonistic muscles is asymmetrical for extending compared with flexing perturbations.

perturbations of comparable velocity with the EMG activities they evoked in biceps and triceps; figure 3.8a. shows the results for the unsupported arm (both antagonists co-contract), whereas figure 3.8b. shows the results for the supported arm (allowing both muscles to silence).
As can be seen, differences were present in the response of the m. biceps to the imposed extensions: in the unsupported situation, the m. biceps responded with an early burst of stretch reflex activity (latency[4] about 30 ms) followed by a silent period (while the muscle was still

[4] All latencies were measured from movement onset. During a perturbation, the first detectable

being stretched!) of roughly 160 ms duration, whereas, with arm support, the m. biceps did not respond during the early phase of the perturbation but displayed two bursts of activity at a later stage of stretching (appr. 190 and 410 ms after perturbation onset, respectively). The

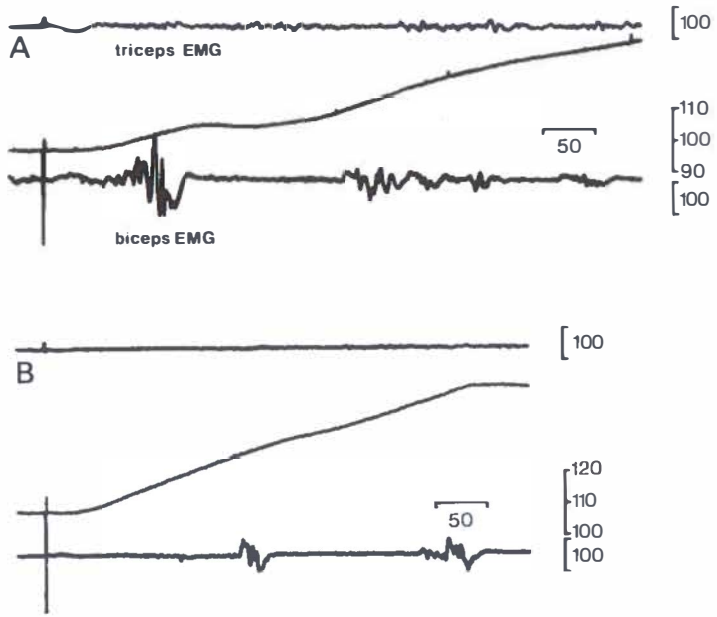

Figure 3.8.:

Raw data on two different perturbations imposed on the arm of one subject (FK). These perturbations were selected on basis of their comparable velocity and amplitude. The experimental conditions were different for the two perturbations: data on the first perturbation (A) were derived without and of the second (B) with support of the arm. The relation was investigated between the timing of EMG activity and changes in torque and calculated δ energy. For each perturbation, the upper trace represents the EMG activity in the m. triceps, the middle trace the position of the arm and the bottom trace the EMG activity recorded from the m. biceps. The biceps EMG during the first perturbation (A) displayed a burst of activity (latency roughly 32 ms after onset of arm movement). The second perturbation (B) displayed some biceps activity but at a later stage of the arm extension (at 194 ms).

movement of the arm (varied with its moment of inertia, but) usually occurred some 40 ms. after clutch activation.

early burst of activity, in the unsupported situation, caused the arm to decelerate and it even tended to move in the opposite direction.

Figure 3.9. displays, for both perturbations of figure 3.8., the data on arm rotation, torque and calculated δ energy plotted as function of time (Figs. 3.9a. to 3.9c.) and as a function of arm rotation (Figs. 3.9d. and 3.9e.). The heavy lines refer to the unsupported situation, the thin

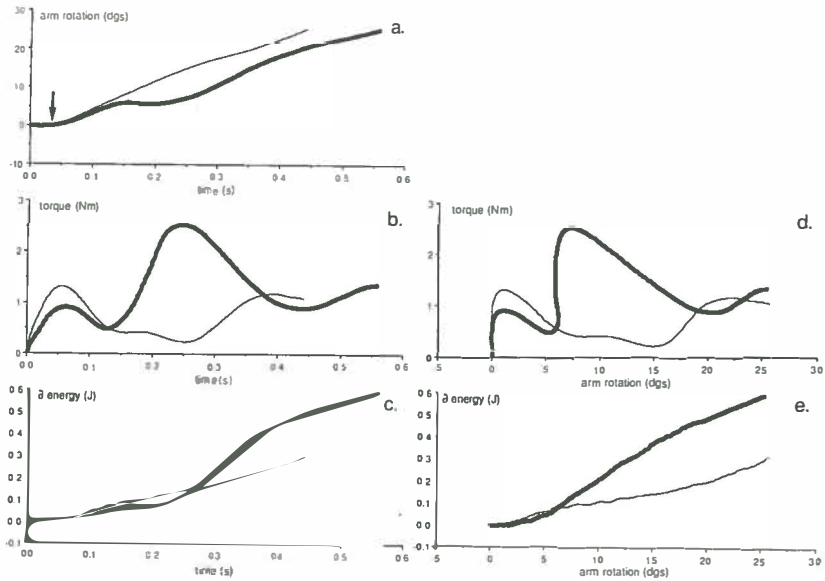

Figure 3.9.:

Data analysis of the perturbations displayed in fig. 3.8: a. replot of the arm position signals of fig. 3.8 (low pass filtered at 67 Hz); b. the torque displayed as a function of time; c. the calculated δ energy displayed as function of time; finally, d. illustrates the torque and e. the δ energy both plotted as function of forearm rotation.
As further detailed in the text, the conclusion of these graphs is that the early stretch reflex activity seen in the EMG of the m. biceps for the first perturbation (heavy line), does not interfere with the analysis of δ energy for the first 100 ms of the perturbation.

lines to the supported arm. A comparison of both experiments shows the effect of the early stretch reflex activity of the m. biceps (Fig. 3.8a.) on the calculated δ energy.

As can be seen from figure 3.9a., the first detectable movement of the arm occurred at about 40 ms. The stretch reflex activity of the m. biceps (heavy line) caused the arm to move in a

direction opposite to that intended by the motor servo some 115 ms after perturbation onset; this caused the motor to impose more torque on the arm as shown in figure 3.9b. The variation of torque with time is complex. It is partly composed by the output of the motor servo and partly by the effects of muscle activity. For instance, the differences in the torque deflection during the

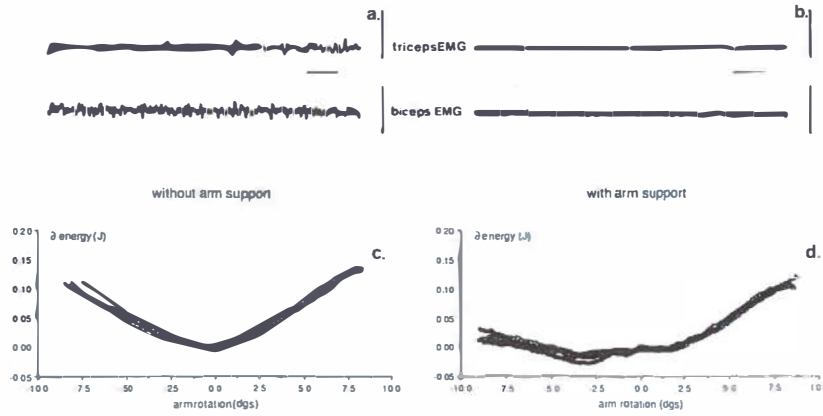

Figure 3.10.:

This plot illustrates the differences in δ energy obtained at different levels of antagonistic co-contraction of biceps and triceps muscle. These differences were obtained for the same subject, instructed in the same way but with both the arm supported and unsupported. With the arm unsupported, the subject maintained some level of antagonistic co-contraction (a), whereas both muscles fell completely silent as soon as arm support was provided (b). The absence in EMG activity in the latter case, caused a marked decrease (d) in the calculated δ energy (the energy absorbed by the elbow muscles) when compared with the condition where antagonistic co-contraction was present (c). These differences were most marked for perturbations in the flexing direction.
Subject: MaB; perturbation amplitude randomized between -30 and 30 dgs; perturbation velocity kept constant at 75 dgs/s.

first 50 ms should be attributed to differences in perturbation velocity aimed at, whereas, the differences in torque arising at 70 to 110 ms (with a maximum at 210 ms after perturbation onset[4]) were the result of the early stretch reflex activity of the m. biceps. When stretch activity was present (heavy line) the torque signal, after its initial deflection, increased so as to overcome the muscular resistance met with, whereas, without muscular activity (thin line) the motor torque decreased after its initial rise.

Figure 3.9d. illustrates how the torque signal is related to the rotation of the arm. On perturbing the arm, early stretch reflex activity in the biceps muscle caused the apparent joint stiffness to increase markedly at about 5 degrees arm rotation (heavy line). Without this stretch activity (thin line), no significant resistance is met with until the arm is extended more than 15 degrees and the effect of the late stretch activity of the m. biceps (Fig. 3.8b., supported arm, delay 195 ms) became apparent.

Figure 3.9e. shows the effect on δ energy of the early stretch reflex activity in the m. biceps. With early stretch activity (thick line) the δ energy is larger than without, but only well outside the critical 100 ms window. Differences emerged at a perturbation amplitude of about 7 degrees arm rotation. Figure 3.9c. shows the variation of δ energy with time: the thick line deviated from its thin counterpart no earlier than at 210 ms after perturbation onset. The marked phase lag of the δ energy is caused by the way it was calculated: by an integration of torque versus arm rotation.

In conclusion, early stretch activity of the m. biceps occurring at a latency[4] of circa 30 ms, led to an increase in motor output torque at a latency of roughly 90 ms and to a gain in δ energy at approximately 210 ms. These findings, studied in detail for two of our subjects, assured us that the δ energy calculated at 100 ms after perturbation onset was free of short latency reflex influences.

The joint stiffness present prior to a perturbation is dependent on the level of antagonistic co-contraction. This is demonstrated by figure 3.10. Differences in EMG activity were introduced by either removing (Figs. 3.10a. and 3.10c.) or providing (Figs. 3.10b. and 3.10d.) arm support to the subject. In the unsupported arm, subjects maintained some EMG activity in extensors as well as flexors (Fig. 3.10a.).

When the forearm was supported, both elbow muscles silenced (Fig. 10b.). Care was taken that removing the arm support did not cause any change in shoulder or elbow position. Also, subjects were instructed as before: they had to sit as relaxed as possible without responding to any movement of the arm. The perturbations were the same for both experiments (fixed velocity of 75 dgs/s; amplitudes randomized in a range of -30 to +30 dgs).

We calculated δ energy for both experimental situations and found an increase in δ energy when the arm was unsupported (Figs. 3.10c. and 3.10d.). For this particular subject, changes in δ energy were most marked for negative perturbations (imposed flexions), though definite differences in δ energy were also present for the imposed extensions.

Finally, figure 3.11. displays a plot of the joint data on δ energy obtained for all ten subjects.

Though there was a considerable amount of scatter, the plot shows a general correspondence in values calculated for the ten subjects. Data on δ energy obtained for the initial stages of perturbations imposed with high velocities were sometimes negative (in four of the ten subjects). We attribute these negative values to an over-estimation of the forearm moment of inertia during the first stages of a rapid perturbation. When forceful jerks were imposed on the arm, the hand occasionally lagged with respect to the moving forearm. In this situation the real

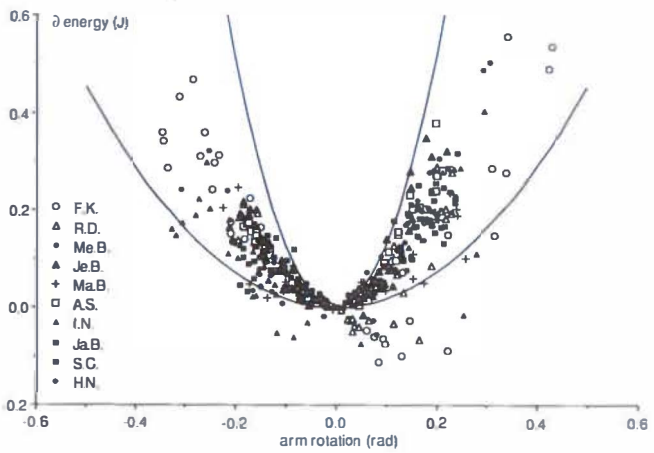

Figure 3.11.:

Plot of the data on δ energy sampled for all ten subjects. Continuous lines represent the 10th and 90th percentiles of the values for joint stiffness calculated from these data. They represent the amount of energy that would be absorbed per radian rotation when the arm could be modeled as a simple two-springs-one-mass system, with stiffnesses of 3.62 for the 10th percentile and 25.77 Nm/rad for the 90th percentile.

moment of inertia of arm and armature was less than expected and less than for low velocity perturbations. This caused an over-estimation of the kinetic energy of the moving arm and armature, and the δ energy became negative. Later during the perturbation these differences disappeared since the hand moved at the same velocity as the forearm and the calculated δ energy returned to values found for less rapid perturbations.

To overcome this problem of unreliable δ energies we applied non-parametric statistics to describe the experimental findings. For each pair of arm rotation (φ) and δ energy (E_δ), we calculated a stiffness value k representing the net stiffness when the arm was treated as a mass

hung between two opposing springs (see Materials and Methods section). For these net stiffnesses we calculated the median value, as well as the 10th and 90th percentiles for each subject separately (Table 3.1.). It appeared that for all subjects 80 percent of the calculated joint stiffnesses fell within a range of 3.62 to 25.77 Nm/rad. The energy absorption appropriate to these two stiffnesses is represented by the continuous lines of figure 3.11.

3.4. DISCUSSION

Most studies on mechanical joint impedance include the action of reflex responses. For instance, Kwan et al. (1979) correlate the presence of medium latency stretch reflexes in the m. triceps with a plateau of joint stiffness, suggesting that this stretch reflex activity is to maintain constant the stiffness of the joint until later muscle actions take over. Somewhat similarly, Nichols and Houk (1976) propose that the function of stretch reflexes, occurring during the first 300 ms of a perturbation, is to compensate for nonlinear muscle properties.

On the other hand, Rack (1970) stresses the importance of the physical properties of muscles for posture maintenance. He shows that, when a muscle is stretched at a low degree of activation, it displays a high initial stiffness which only lasts for a limited amount of stretch. Rack argues that the passive properties of a muscle are more important for instantaneous responses to muscle stretch than are spinal stretch reflexes: during the initial phases of a perturbation the muscle stands alone in resisting a sudden perturbation. The initial resistance to a perturbation can only be influenced by the CNS through changes in the level of antagonistic co-contraction.

The present investigation aimed to measure the mechanical impedance of the elbow joint and its dependence on antagonistic muscle action. Perturbations were imposed on the forearm of ten volunteers. Perturbing torques and resulting arm rotations were recorded only for the first 100 ms of each perturbation since we aimed to calculate the limb resistance against an imposed perturbation excluding early reflex activity. The figure of 100 ms was derived from the latency of the myotatic stretch reflex of m. biceps (c 18 ms) added to the delay required to generate a force out of electrical muscle activity (estimated at 80 ms). Hagbarth, Hägglund, Wallin and Young (1981), for wrist extensors, give figures of 40-60 ms before the electrical activity causes measurable muscle torque, attaining its peak some 40 ms later. Hammond et al. (1956) show that EMG responses to a sudden stretch of the m. biceps do not result in muscle force until after

some 90 ms, with mean latencies exceeding 100 ms. Figures 8 and 9 support our assumption that stiffness analyses based upon the first 100 ms of a perturbation do not contain the effects of early reflex activity.

The mechanical joint impedance is the net opposing torque exerted by the forearm against an imposed perturbation. The mechanical joint impedance arises from: (1) the sudden acceleration of the mass of the forearm and (2) the visco-elastic properties of the muscle groups (and other soft tissues) crossing the elbow joint. The analysis proposed in the present study makes it possible to determine the relative contribution of either of these two mechanisms to the overall mechanical joint impedance.

If mechanical joint impedance were to be analyzed within the torque domain, a signal of arm acceleration would be required. This could be extracted from the position signal by taking its second derivative. It appeared, however, that the acceleration signal obtained in this way was blurred by the inevitable amplification of the goniometer output noise (compare Kwan, Murphy and Repeck, 1978). Therefore, the analysis was performed in the energy domain. We calculated the amount of work (input energy; Fig. 3.5d.) exerted by the motor in perturbing the arm and subtracted the energy required for the acceleration of the forearm (figure 5f). In this way, we obtained the energy (Fig. 3.5h.) absorbed by the antagonistic muscles crossing the joint (and due to the visco-elasticity of other structures, such as the joint capsule).

The plot of input energy versus arm rotation (Fig. 3.5d.) can also be taken as an illustration of an energy landscape in which the subject's arm lies: the more energy that is required to move the forearm, the more stable it is held in position. However, we calculated that depending on perturbation velocity 2 to 89 % of the input energy is utilized to accelerate the arm. This is in contrast to Humphrey and Reed (1983) who calculate, for the monkey wrist, a contribution to the total stiffness of 5-10 % arising from the moment of inertia of the hand and from joint and tissue visco-elasticity, and of 90-95 % resulting from muscle elasticity and antagonistic co-contraction. However, these data were obtained for the alert monkey which actively resisted perturbation torques varying sinusoidally with an amplitude of 98 Nm. The present study investigated the stiffness of the human elbow joint, while subjects were instructed not to respond to a movement of the arm. Subjects, therefore, did not deliberately co-contract their elbow muscles.

If we regard the posture of the elbow joint as the result of a local minimum in an energy landscape, the steepness of this energy minimum is mainly dependent on the moment of inertia of the forearm and only to a lesser extent on the visco-elasticity of the elbow muscles. This

indicates that it is unlikely for the arm to start moving spontaneously at a high velocity. However, it also suggests that slow postural drift is less well antagonized.

For movements at low velocity, the mechanical joint impedance collapses to the joint stiffness illustrated by figure 3.5h. Consequently, prevention of slow drift depends entirely upon the co-contraction of elbow antagonists: if the forearm were not stabilized by the elbow muscles it would behave as the dummy load illustrated in figure 3.5g. Therefore, it was of interest to investigate with what stiffness the forearm was held in place by our subjects: we obtained 10th and 90th percentiles of 3.62 and 25.77 Nm/rad for the ten subjects studied.

Again, it should be emphasized that the calculated values representing joint stiffness remain partially derived from velocity dependent muscle forces: when a muscle is being stretched, the exerted tension becomes almost twice that recorded for isometric conditions (Aubert, 1956; Otten, 1987), and, if a muscle is shortened at a speed higher than its maximal contraction velocity (V_{max}), its tension will drop to zero (Hill, 1938). As muscle lengths changed significantly during the first 100 ms of a perturbation, the contribution to the (apparent) joint stiffness of velocity dependent muscle force cannot be excluded.

In contrast to MacKay et al. (1986), the present study has made no attempt to differentiate between the contribution of length and velocity dependent muscle force to overall joint stiffness. In order to make such a differentiation, MacKay et al., however, assume linear muscle visco-elastic properties as well as a symmetrical arrangement of antagonistic force-length characteristics. Under these assumptions, MacKay et al. simulate their experimental data by means of a computer model, obtaining joint stiffnesses ranging from 1.0 to 12 Nm/rad at 90 degrees elbow flexion. These data are slightly less than those obtained in the present study, possibly because MacKay et al. study the passive arm, whereas the present paper studied joint stiffness with a moderate level of antagonistic co-contraction present.

Finally, figure 3.10. shows how the elbow joint stiffness decreased when both antagonistic muscles were allowed to silence by supporting the forearm against gravity. This is a simple demonstration that antagonistic co-contraction increases the stiffness of the joint (Humphrey and Reed, 1983). The presence or absence of an arm support cannot explain these differences in observed joint stiffness: arm support was provided against a lowering of the forearm in the vertical direction, while all stiffness analyses were performed on movements in the horizontal plane.

Holding the forearm up against gravity required an exorotation of the shoulder. (Interestingly, after the full experimental session, subjects usually complained about fatigue in the dorsal part

of their m. deltoideus, indicating that this muscle section was primarily responsible for shoulder exorotation.) Since the mm. biceps and triceps only have a minimal exorotating momentum, removal of the arm support by itself did not necessitate activation of these muscles. Yet, both biceps and triceps displayed electrical activity as soon as the arm support was removed. Possibly, a general sense of arm instability causes the elbow muscles to co-contract.

Conclusion

The present investigation has shown that elbow joint posture is maintained by a balanced tuning of antagonistic muscle groups, resulting in a local minimum in an energy landscape in which the arm lies. The steepness of this energy minimum depends upon (1) the moment of inertia of the limb and (2) the visco-elasticity of the muscles (and other soft tissues) at the elbow joint. The contribution of the first to the overall mechanical joint impedance is large for high velocities of the forearm, but becomes negligible for low arm velocities. Consequently, rapid changes in arm posture are well prevented by the forearm's moment of inertia but slow drift in elbow joint posture is fully dependent on the joint visco-elasticity originating from the co-contraction of antagonistic muscle groups.

Of joint visco-elasticity, it is the elastic component (or joint stiffness) which is responsible for the prevention of slow postural drift. Crucial is that the energy minimum set up by joint stiffness is kept fixed at the angle to be maintained. This necessitates a constancy of antagonistic muscle activations over a long period of time (up to minutes).

We suggest that the myotatic stretch reflex is of major importance for an accurate tuning of antagonistic muscle tone. This reflex arc derives its activity from muscle spindles which are renowned for their high sensitivity to small changes in length. It has been shown experimentally that the high spindle sensitivity to stretch is based on a fixation of groups of intrafusal cross-bridges, a mechanism often called stiction (Matthews, 1972; Goodwin et al., 1975; Proske and Stuart, 1985; Hunt, 1990). Fixed intrafusal actin-myosin bonds cause a stretch at the spindle poles to be transmitted directly to the sensory region of the intrafusal fibres. The high length sensitivity (up to 280 pps/mm: Hasan and Houk, 1975) holds for stretches up to an amplitude of 25 μm spindle length. For the elbow joint this compares to a rotation of approximately 2 degrees[5].

[5] As the fibre length of the m. biceps amounts nearly 150 mm (An, Hui, Morrey, Linscheid and Chao, 1981) and the length of a muscle spindle is about 10 mm, a 25 μm stretch of the muscle spindle would correspond to a 0.4 mm stretch at the level of the m. biceps. As the working arm of the m. biceps at an elbow angle of 80 degrees is about 14 mm (An et al., 1981), the

Stable actin-myosin bonds form spontaneously at any given spindle length (Matthews, 1972; Goodwin et al., 1975; Morgan, Prochazka and Proske, 1984; Gregory, Mark, Morgan, Patak, Polus and Proske, 1990). Thus, when a muscle is kept at constant length for some period of time, the high length sensitivity of its muscle spindles spontaneously re-establishes, centred at the prevailing spindle length. In this way, stiction occurring in intrafusal fibres may establish an absolute control of antagonistic muscle length at any given elbow angle. Thereby, the minimum in δ energy keeping the forearm in place is prevented from drifting away. The only risk of the described control system is that it may develop oscillations if the feedback gain is too high[6]. Probably, the system is prevented from these oscillations by the velocity sensitivity (and phase advance) of the muscle spindle response (Crowe and Matthews, 1964; Brown, Engberg and Matthews, 1967).[1]

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corresponding change in elbow angle would amount $\sin^{-1}$ (0.4 / 14) = 2 degrees.

[6] Interestingly, it is generally assumed that the gain of the myotatic stretch reflex is too small to be able to correct for changes in length or in load. This assumption is derived from the calculation Matthews (1966) makes on basis of his measurements on the reflex increase in muscle force obtained in response to sinusoidal stretching (tonic vibration reflex). Matthews calculates a maximal increase in muscle force of 30 g wt. in response to a muscle stretch of 1 mm, which is less that 2 % of the maximal isometric soleus force. This calculation assumes a muscle spindle sensitivity of 10 pps/mm stretch. However, Hasan and Houk (1975) show that the spindle sensitivity may amount as much as 280 pps/mm, provided that the spindle remains in its linear range. This would lead to an increase in muscle force per mm soleus stretch of more than 50 % of maximal.

CHAPTER 4

Postural rigidity, elasticity and plasticity.

4.1. INTRODUCTION

One of the early papers studying in detail EMG responses to an imposed perturbation of the arm, is that of Hammond, Merton and Sutton (1956). In their experimental paradigm subjects have to resist a pre-load by contraction of the m. biceps. Suddenly, the arm is pulled away, and Hammond et al. describe the changes in EMG activity occurring in response to the imposed stretch of the m. biceps. Already, these authors emphasize how a subject's response depends on the instruction given prior to the experiment.

EMG responses to a suddenly imposed stretch have been subdivided in (review: Marsden, Rothwell and Day, 1983): 1. short latency reflexes occurring at spinal latencies of 15-30 ms (for proximal arm muscles); 2. (medium and) long latency reflexes occurring at latencies of 40-70 ms (called 'automatic as these latencies were assumed to be too long to be spinal and too short to be voluntary); and 3. 'voluntary' responses occurring at latencies larger than ± 80 ms. This subdivision is used by many other investigators studying EMG responses to perturbations imposed on various parts of the body (on the thumb: Marsden, Merton and Morton, 1972; on the forearm: Crago, Houk and Hasan, 1976; Kwan, Murphy and Repeck, 1979; Ma and Zahalak, 1985; on the whole body: Nashner, 1976; Marsden, Merton and Morton, 1981). It suggests that voluntary responses may be distinguished from automatic and spinal responses by means of their latency. However, not all EMG responses occurring at latencies exceeding 100 ms need to be voluntary (in the sense that they require a subject's alert attention).

All studies mentioned above have in common that they record EMG responses supposedly resisting an imposed change of limb (or body) posture. That a subject gives this type of response is motivated either by his instruction ('maintain a steady posture as well as you can') or by the nature of the task itself (as in the case of Nashner's experiments). Many investigators (e.g. Crago et al., 1976; Marsden et al., 1983) report that as soon as a different instruction is given ('let go when a perturbation is imposed'), or when experimental conditions are altered so

that long latency responses provide an undesirable effect, these responses can be modified rapidly. Nashner (1976), for instance, shows that long latency reflexes occurring in muscles of the leg are suppressed within 2 to 3 trials, when they elicit unwanted muscle contractions in response to an imposed perturbation. Marsden et al. (1981) show that different EMG activities are recorded from muscles of the arm in response to a sudden postural sway, depending on whether a subject's hand has hold of a stable object (such as the side of a table) or whether it is, for instance, balancing a cup of tea.

Several investigators (Asatryan and Fel'dman, 1965; Gottlieb and Agarwal, 1988; Latash and Gottlieb, 1990) study the behaviour of a subject's arm in response to step changes in load. At the start of these investigations, subjects resist a pre-load which is then, unexpectedly, either increased or decreased. Subjects are instructed 'not to react' and the step change in load causes the forearm to move, with some oscillation, to a new position. The first investigations of this kind (e.g. Asatryan and Fel'dman, 1965) report that, after a change in load, no changes are observed in EMGs recorded from the elbow muscles. Therefore, it seems reasonable to take the final angle the elbow joint reaches after a change in torque, as a measurement of muscle stiffness, the muscle activation being the same as that required to maintain the initial joint angle. This, however, proved too simple an assumption, since it is shown by later studies that changes in EMG do occur after a step change in torque (Latash and Gottlieb, 1990).

The present study was motivated by a simple observation; that compliant behaviour of the human elbow joint does not only occur after a step change in torque, but also after a pulse in torque. If the forearm of a subject is rotated by an external force of limited duration (for instance, by an examiner) it can be seen to remain in a new posture, even when this force is discontinued, provided that the subject is instructed not to respond. This observation suggests that a passive[1] movement of the arm may induce changes in EMG activity which subserve postural plasticity. It was though of interest to investigate this mechanism, as postural plasticity may counteract the concept that posture is rigidly maintained by the action of myotatic reflexes, implicitly suggested by the many reports on increasing EMG activities in response to muscle stretch.

The present study proposes that plasticity is as important as rigidity for posture maintenance at the human elbow joint. It investigates whether postural plasticity can really be demonstrated and whether it arises from passive properties such as, for instance, thixotropy (Lakie, Walsh and

[1] The term 'passive movement' is used in the clinical sense and designates the movement of the arm by an external force.

Wright, 1984; Hagbarth, Hägglund, Nordin and Wallin, 1985) or from the active control by the central nervous system (CNS) of antagonistic muscle activation (as suggested by Sherrington, 1909). For this purpose, EMG activity of the mm. biceps and triceps as well as changes in maintained elbow joint angle were studied in response to various pulses in torque imposed on the forearm.

4.2. MATERIALS AND METHODS

As in our previous paper (Schaafsma and Van Willigen, 1991), this study describes the results of perturbations imposed to the forearm of ten healthy human volunteers (aged 26 to 52, two females and eight males). All subjects gave their informed consent and, except for one of the authors, were unaware of the hypotheses tested. The experimental procedures were approved by the local ethical committee.
We used the same perturbation protocol as described previously; this section will only deal with techniques which have not yet been described.

EMG recordings as function of joint angle
To relate antagonistic muscle activity with elbow joint posture, EMG activities of the mm. biceps and triceps were sampled for different angles of the joint. We mounted each subject in the perturbation apparatus described earlier (Schaafsma and Van Willigen, 1991). The subject's arm was unsupported, so that the mm. biceps and triceps maintained some basic level of electrical activity. We asked each subject to hold, as relaxed as possible, his (or her) arm at a particular joint angle. When the subject had his (her) arm completely relaxed in this position, (s)he gave a sign to the experimentator who triggered the data sampling. In the domain of 40 to 180 degrees joint angle, for 25 trials in a series and in a non-systematic order, subjects were asked to chose a different joint angle as soon as sampling was completed.
The computer sampled data on elbow joint angle (derived from the goniometer) and on the EMG activity of both antagonistic muscles (duration 0.5 s, frequency 9.6 kHz per channel). Data were processed immediately: an average value was calculated from the position signal, providing the elbow angle; digitized EMGs were first processed by removing their DC level and by then rectifying them, before an average value was calculated over the full sampling period (0.5s), giving one single value for the total amount of EMG activity in the biceps and triceps muscle, respectively.

EMG recordings during imposed perturbations

As before, EMG activities of the mm. biceps and triceps were recorded during successive perturbations and stored on hard disc. Perturbations were randomized with respect to their velocity (ranging from ±20 dgs/s to ±150 dgs/s) and to their amplitude (ranging from -30 to 30 degrees). Unless stated otherwise, all subjects were instructed not to react to movements of their arm.

In three of our subjects we made a detailed study of EMG responses to a perturbation of the arm (velocity 150 dgs/s; clutch activation of 0.02 or 0.20 s) when the arm was supported, unsupported or when the instruction of the subject was changed from 'do not respond' to 'maintain the arm in the same posture as well as possible' (this last set of experiments was performed with the arm unsupported; visual feedback was allowed). The recordings obtained from one of these subjects, are illustrated in the present paper.

Determining the median plasticity ratio

During each perturbation, data were stored on hard disc for later retrieval and analysis. A schematic representation of an imposed perturbation is provided by figure 4.1., in order to show what data were sampled for computer analysis (see legend).

It was studied whether, after an imposed perturbation, the arm returned to its starting position or not. For that, we determined the angle offset, which was the difference between final and starting position of the arm (Fig. 4.1.: 8 - 5). We plotted this angle offset against the maximum perturbation amplitude: the difference between the maximum amplitude attained by the arm after the perturbation minus its starting position (Fig. 4.1.: 7 - 5). We introduced a plasticity ratio, which was defined by the ratio of angle offset divided by maximum perturbation amplitude. Of the full series of perturbations imposed on a subject's arm we determined the median plasticity ratio as a figure of the subject's general plastic behaviour.

Determining the summed reflex score

All subjects were screened on the following clinical reflexes: biceps reflex (score 0, 1/2, 1, 2, 3, 4, 5; >3 pathological); triceps reflex (score 0, 1/2, 1, 2, 3, 4, 5; >3: pathological), brachialis reflex (score 0, 1/2, 1, 2, 3, 4, 5; >3 pathological), Mayer reflex (score 0, 1/2, 1) and Hoffman-Trömner reflex (score 0, 1/2, 1). Biceps and triceps reflexes were obtained by percussion of the tendons by means of a tendon hammer, causing a reflex flexion or extension of the arm. The brachialis-reflex was obtained by percussion of the distal part of the radius

while the arm was supinated and 90 degrees flexed at the elbow. The reflex was scored by the extent to which it evoked a pronation of the forearm. The Mayer reflex was scored by the

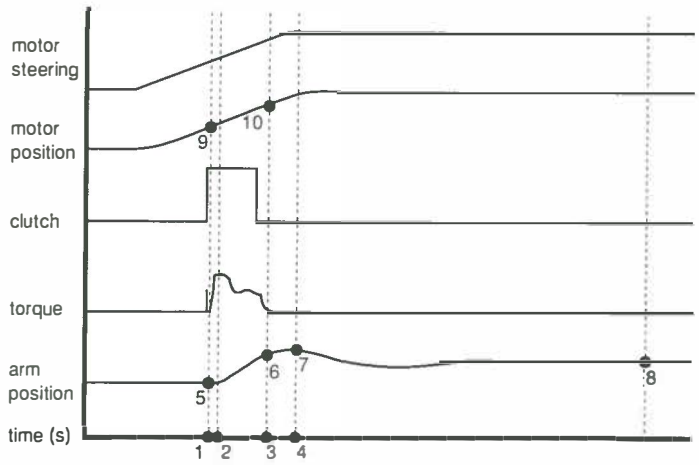

Figure 4.1.:

Schematic representation of an imposed arm perturbation. Measurements taken from the data stored on hard disc were indicated with filled circles and numbered according to the following description: after the motor was brought into motion the clutch was activated with a brief pulse. There was a short delay between the activation of the clutch and the first measurable exertion of torque (no. 1) as well as between the inactivation of the clutch and the diminution of exerted torque (no. 3). The sudden transmission of motor torque through the clutch caused an abrupt perturbation of the starting position of the forearm (no. 5). The first instance for which a detectable change in arm position could be measured indicated the actual start of the perturbation (no. 2). The position of the arm at disappearance of the exerted motor torque (no. 6) was used for the determination of the actual perturbation amplitude. After the clutch had fully detached, the arm could move freely, and due to its inertia it usually swung somewhat further even after the motor torque had been discontinued. In this way it reached a maximum amplitude (no. 7) of which the time of occurrence was also registered (no. 4). The final position at which the arm came to a rest (no. 8) indicated to what extent the arm position had successfully been restored. Finally, two recordings of the motor position, at the onset and at the end of each perturbation (nos. 9 and 10) served as control whether the motor velocity aimed at was actually achieved.

amount of opposition of the thumb in response to a forceful flexion of the most proximal phalanx of the middle finger. Finally, the Hoffman-Trömner was elicited by lifting the relaxed

hand of the subject and tapping against the palmar surface of the most distal phalanx of the middle finger. The amount of flexion in the remaining fingers of the hand determined the score for this reflex.
Scoring of reflexes was performed by the same investigator for all ten subjects and was based on the most vivid response that was obtained. Reflexes were evoked bilaterally and a final score was calculated by adding the scores on the five reflexes for both sides. By definition, for normal subjects a score was expected no larger than twenty-two (the maximal score was thirty-four).

4.3. RESULTS

If one regards a maintained angle of the elbow joint as the result of differences in antagonistic co-contraction, it is of interest to determine how the EMG activities recorded from the mm. biceps and triceps correspond with elbow joint angle. This is shown by figure 4.2.
Rectified and averaged EMG activities recorded from the mm. biceps and triceps are displayed while the subject maintains, as relaxed as possible, different joint angles of the elbow joint. It is demonstrated that, the larger the joint angle, the greater the required activity of the m. triceps and the smaller the activity observed in the m. biceps. The more the arm is held in a flexed posture, the more the biceps becomes active and the more the m. triceps becomes electrically silent. Though over the full range of elbow joint angles a clear picture emerges about the relation between antagonistic muscle activation and elbow joint angle, such a relation becomes less conspicuous when we regard smaller changes in joint angle: there is a considerable scatter in the data on EMG activity.
It should be noted that tonic EMG activities recorded in these experiments were generally very small: in the range close to 90 degrees flexion, peak-to-peak amplitudes of raw EMGs were rarely greater than 50 μV. Such activities were less than 1 percent of the peak-to-peak amplitudes recorded for maximal isometric forces (with identical electrode configuration).

With the relation between differences in antagonistic muscle activation and elbow joint angle established, changes in EMG activity were studied in response to perturbations imposed on the forearm. In three of the ten subjects it was investigated how responses to a perturbation depended on whether the arm was supported or not and how they depended on the instruction

given to the subject prior to testing. The results obtained from one subject are displayed in figure 4.3.

Figure 4.3a. shows the response to a perturbation when the subject's arm was supported. Providing support to the arm caused the elbow antagonists to display less electrical activity than without arm support, in some subjects (not illustrated) both muscles even silenced completely. In response to a perturbation, very little change in EMG activity is observed. From its starting position at 95 dgs, the arm was flexed to an elbow angle of 42 dgs (maximal perturbation

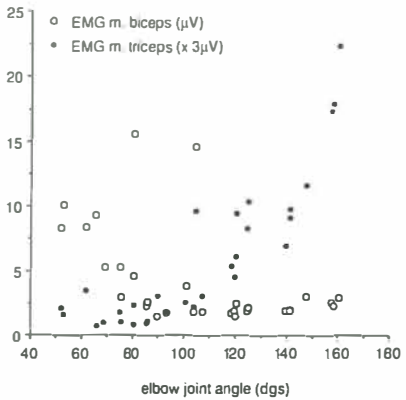

Figure 4.2.:

Plot illustrating the relation between elbow joint posture and rectified and averaged EMG activities of the mm. biceps and triceps, respectively (Subject: AS). An increasing joint angle corresponds with a decrease in the activity of the m. biceps and an increase in the activity of the m. triceps.

amplitude: -53 dgs). After the perturbation, the arm trajectory displayed a low damped oscillation and came to rest at 96 dgs. For this perturbation a plasticity ratio was calculated of 2%.

Figure 4.3b. shows the response of this subject to the same perturbation, but now without arm support. The resulting instability of the forearm caused a slight increase in the level of antagonistic co-contraction. Moreover, the response to an imposed perturbation became different: instead of the absence of changes in EMG observed for the supported arm, the

unsupported arm responded with a burst of activity in the m. triceps (latency[2] circa 30 ms); and with a marked increase in activity in the shortening m. biceps (latency 260 ms).

So, in response to a perturbation, not only the stretched muscle, but also the shortening muscle may increase its EMG activity. The increase in biceps activity was maintained for the full length of the experiment and seemed responsible for the permanent change in maintained elbow angle: from its starting position of 90 dgs, the arm was perturbed with a maximal amplitude of -51 dgs, and, after a damped oscillation, stabilized at a final angle of 76 dgs. The plasticity ratio of this perturbation amounted to 27%.

Comparing figures 4.3a. and 4.3b., it can be concluded that removing the arm support caused: (1) an increase in antagonistic co-contraction; (2) an increased damping of the arm trajectory after a perturbation and (3) an increase in postural plasticity.

Figure 4.3c., finally, shows the subject's response when instructed to maintain, with visual feedback, the starting position of the arm as well as possible. Under these circumstances, the arm trajectory did not display the low damped oscillation as observed in figures 4.3a. and 4.3b.; its trajectory was angular, as the forearm started moving back to its starting position as soon as the clutch was released. The EMG of the stretched m. triceps showed a burst of activity (note that the vertical calibration mark is two times smaller than for figures 4.3a. and 4.3b.). Also, the biceps activity increased in response to the perturbation, some 45 ms after perturbation onset. The marked activity in both muscles did not result in a very accurate repositioning of the arm: from its starting position of 102 dgs, the arm was perturbed with a maximal amplitude of -16 dgs and stabilized at 100 dgs (the plasticity ratio was 12.5%).

The increase in triceps activity occurred as soon as 14 ms after the first detectable arm movement, which is about the latency of a triceps tendon jerk. As short latency activity was low (or absent) in the previous two experiments when the subject was instructed not to respond, this suggests that a subject can, by anticipation, increase the output of the spinal stretch reflex.

The experiment of figure 4.3. demonstrated that marked differences could be obtained in response to identical perturbations, depending on the conditions of the arm (supported or not) and on the instruction of the subject. For the remainder of this paper we studied responses to imposed perturbations while a subject's arm was not supported and under the instruction not to react to the movements imposed.

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[2] All latencies were calculated with respect to the instant of perturbation onset. The start of arm movement was determined from high resolution plots of the arm positions signal. Usually, the arm started moving some 40 ms after activation of the clutch. This latency depends on the gain of the motor servo, correcting the motor position when it deviates from the trajectory aimed at.

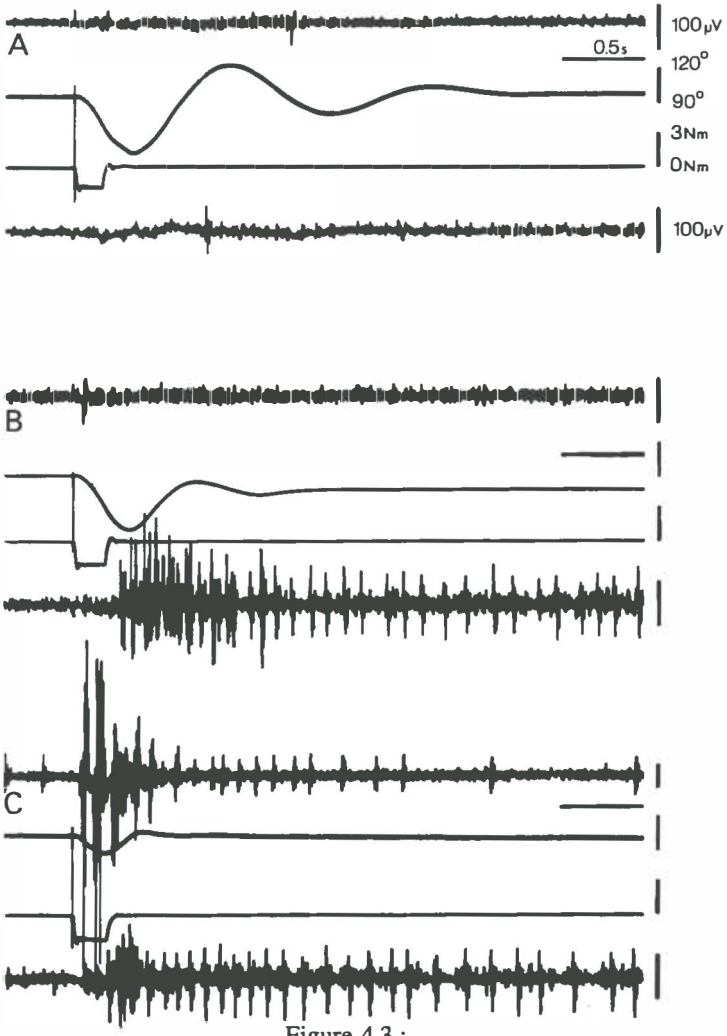
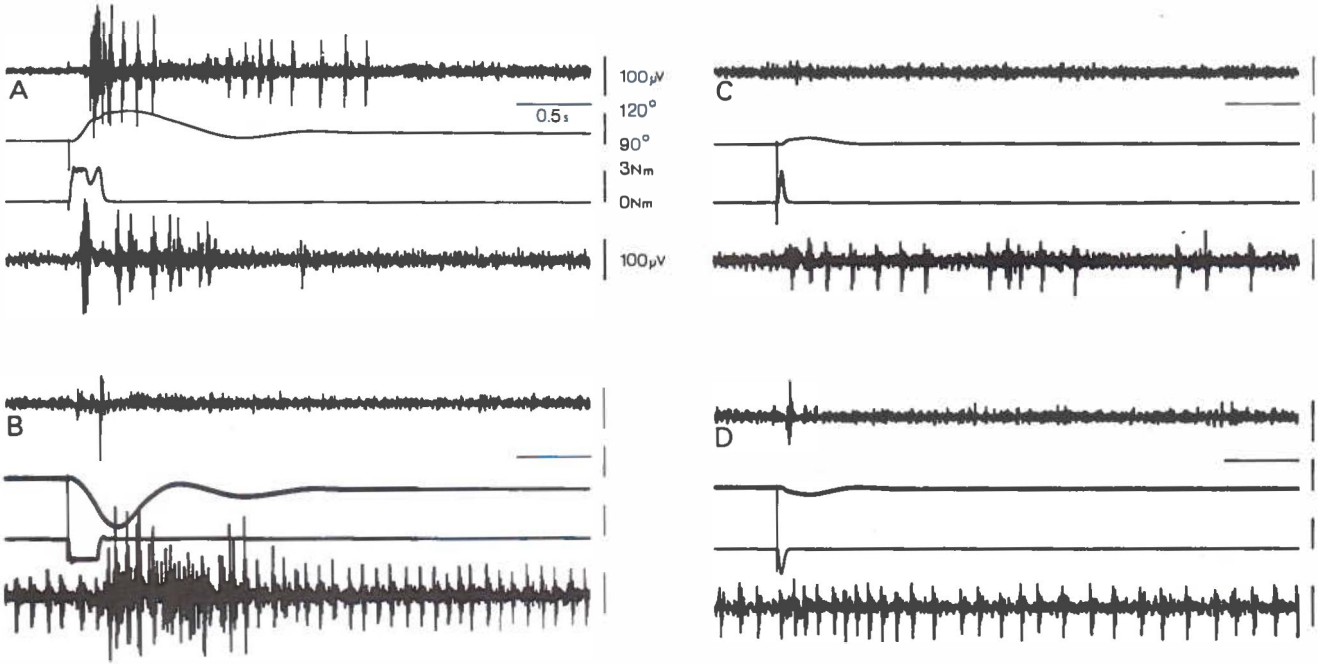


Figure 4.3.:

Data on three perturbations imposed on the arm of a single subject (FK). Each perturbation is displayed by four traces: triceps EMG being displayed by the top trace, elbow joint angle by the second, motor output torque by the third and biceps EMG by the fourth and bottom trace. For all traces, vertical calibrations correspond with 100 μV for the EMGs (note smaller calibration bar for triceps EMG in C), with 30 dgs for elbow angle and with 3 Nm for motor torque; horizontal calibrations correspond with 500 ms.
The conditions for the three perturbations differed: in A. the subject's arm was supported; in B. the arm support was removed; and in C. the subject was instructed to maintain constant, as well as he could, the posture of the unsupported arm. (for discussion see text).


A
0.5s
100μV
120°
90°
3Nm
0Nm
100μV
B
C
D

← Figure 4.4.:

Data on four perturbations imposed on the arm of a subject (FK), showing the differences in response to a large extension (A), to a large flexion (B), to a small extension (C) and to a small flexion (D). It is demonstrated that after a small perturbation the arm returns to its starting position with little change in EMG activity. After a large perturbation, changes in EMG activity are observed, which, however, do not result in a return of the arm to its starting posture: the arm behaves plastically. Display and calibrations as in figure 4.3.

Figure 4.4. illustrates the subject's responses as function of the amplitude of an imposed perturbation. The differences in perturbation amplitude were obtained by varying the duration of clutch activation at a constant motor velocity: in figures 4.4a. and 4.4b., the clutch was activated for 0.20 s; in figures 4.4c. and 4.4d. for 0.02 s. The motor velocity was ±150 dgs/s, depending on whether the arm was flexed or extended.

Figure 4.4a., which illustrates the subject's response to an imposed extension (maximal amplitude +31 dgs), shows a burst of EMG in the m. biceps (latency 34 ms). Also, the m. triceps responds with an increase in activity, while the muscle is still shortening (latency 113 ms). Considering the tonic level of activation maintained after the perturbation, in this experiment a marked increase in tonic EMG activity was observed in both the m. biceps and triceps. However, in most experiments, the lengthening muscle (the m. biceps) responded with a decreased or constant tonic EMG activity. Apparently, the increase in biceps activity did not outweigh the increased triceps activity as the forearm shifted permanently to a more extended position (from a starting position of 91 dgs to a final angle of 100 dgs; giving a plasticity ratio of 29%).

Figure 4.4b., on the other hand, displays the subject's response to an imposed flexion of the arm (maximal amplitude -50 dgs). This flexion induced very little stretch activity in the m. triceps (latency 24 ms). Instead, a marked increase was observed in biceps EMG activity (latency 235 ms) comparable to that seen in figure 4.3b. It was hard to tell whether the tonic EMG activity in the m. biceps had increased after the perturbation: a large unit[3] which, apparently, was close to the surface electrodes, obscured the tonic level of biceps activity. The

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[3] On the basis of figure 4.4d. alone, it is hard to be sure that the observed periodic EMG activity in the m. biceps was indeed derived from a single motor unit, the more because its amplitude was not quite constant. However, in a plot on an expanded time scale it was ascertained that the configuration of this periodic EMG activity was quite constant throughout the experiment, suggesting that, it was indeed derived from a single motor unit firing at about 10 Hz.

discharge frequency of the unit increased slightly in response to the perturbation (from 9.0 to 10.5 Hz). Nevertheless, it can hardly be expected that only the increase in frequency of this single unit can fully explain the permanent shift of maintained elbow posture: after the imposed movement the arm settled at a position of 78 dgs, which was 12 dgs more flexed than its starting position of 90 dgs. The plasticity ratio was 24%.

Characteristically, the EMG activities of the mm. biceps and triceps in response to a small perturbation were much less than in response to a large. Figure 4.4c. shows the subject's response to an imposed extension of 7 dgs. Only a small burst of stretch activity was present in the biceps EMG (latency 31 ms). There was some irregular firing of a unit close to the surface electrodes. At crude inspection, the tonic EMG activity in the m. triceps as well as in the m. biceps remained the same, irrespective of the imposed perturbation. The arm returned to a position of 89.9 dgs, after being perturbed with a maximal amplitude of +7.2 dgs from its starting position at 89.0 dgs. The calculated plasticity ratio was 12.5 %.

The plasticity ratio was less for the small imposed flexion of figure 4.4d.. In this experiment, the plasticity ratio amounted 1.4% (the arm was perturbed with -7.3 dgs from its starting position of 90.8 dgs and, after the perturbation, settled at a position of 90.7 dgs). During this experiment, some stretch activity was observed in the m. triceps (latency 32 ms), as well as a temporary increase in the discharge of the single motor unit recorded in the two previous experiments. Again, very little tonic changes were observed in the level of antagonistic EMG activity.

It appeared that the behaviour pictured in figure 4.4. is not constant throughout an experimental session. This is illustrated in figure 4.5., which displays a subject's response to perturbations, imposed with identical parameters as in figure 4.4a. They were selected out of a series of randomly imposed perturbations to investigate the extent to which the subject's response was reproducible.

In all three figures a similar trajectory of the arm can be observed as in figure 4.4a. Nevertheless, the calculated plasticity ratio differed from one experiment to another: for figure 4.4a. the plasticity ratio amounted 29% and for figures 4.5a. to 4.5c. it amounted 16, 9 and 26%, respectively.

The changes in EMG activity also varied from trial to trial. In figure 4.4a. there was an increase in the tonic EMG of the mm. triceps and biceps. A tonic increase in triceps EMG is also found in figures 4.5a. and 4.5c., but seems low or even absent in figure 4.5b. The tonic increase in biceps EMG observed in figure 4.4a. is absent in figure 4.5a., 4.5b. as well as 4.5c. The

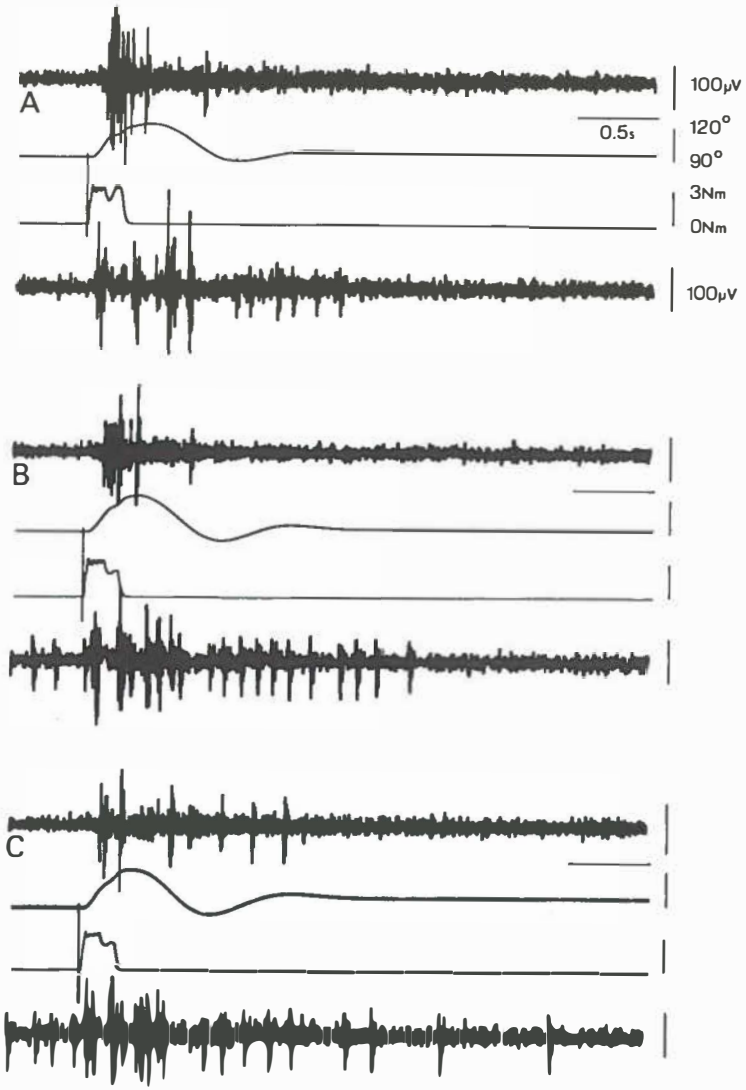

Figure 4.5..

Data on three identical perturbations imposed on the arm of a subject (FK), showing the variability in EMG response as well as in final arm posture. Though there is a general similarity in EMG response between different trials, they differ when inspected in detail. Also, the shift in arm posture (postural plasticity), varies slightly from trial to trial. Display and calibrations as in figure 4.3. and 4.4.

disappearance of a tonically active motor unit present (prior to the perturbation) in figures 4.5b. and 4.5c., even suggests a lowering of biceps activity after muscle stretch.

In general, this series of experiments shows that, apart from the expected increase in EMG activity of the stretched muscle, a marked increase is often seen in the EMG activity of the shortening muscle. Also, the degree of restoration of the starting position was greater after small than after large amplitudes of perturbation. The relatively accurate return of the elbow posture after a small perturbation is accompanied by an absence of tonic changes in EMG, whereas the permanent shift in maintained elbow angle after a large perturbation is often found to correspond with a tonic increase of activity in the shortened muscle (and/or a tonic decrease of activity in the lengthened muscle).

Apart from differences in behaviour within one subject, we also observed differences between subjects: for the ten subjects studied the behaviour in response to a comparable set of perturbations varied one from another. An example of inter-individual variation is provided by figure 4.6.

In figure 4.6a. the response of a subject is shown to a large imposed flexion (maximal perturbation amplitude -35 dgs). Despite the fact that the subject's arm was unsupported, this subject displayed very little EMG activity in the antagonistic muscles of the elbow prior to the perturbation. Moreover, no EMG responses occurred during and after the imposed flexion of the arm. The subject's arm trajectory showed a low damped oscillation, and the final arm position differed only little from the one prior to the perturbation (plasticity ratio 2%).

In contrast, figure 4.6b. shows the response of a second subject. This subject did show a co-contraction of antagonistic muscles prior to the perturbation. In response to a similar imposed flexion (maximal perturbation amplitude 31 dgs), this subject responded with a small, phasic, increase in triceps activity (latency 71 ms) and a prolonged increase in biceps EMG activity (latency 97 ms). This increased tonic activity of the m. biceps caused a permanent shift in elbow angle (plasticity ratio 84%).

The behaviour illustrated in figure 4.6a was characterized as 'elastic'; that illustrated in figure 4.6b. as 'plastic'. During an experimental session it was never observed that one subject responded elastically on one occasion and responded plastically on another. This is shown by figure 4.7., which illustrates that the behaviour of both subjects of figure 4.6. was consistent throughout the experimental session. Arm trajectories, in response to a series of randomized perturbations, were plotted in one figure. Figure 4.7a. shows arm trajectories obtained after perturbations imposed on the 'elastic type'. This subject responded with a stereotypical

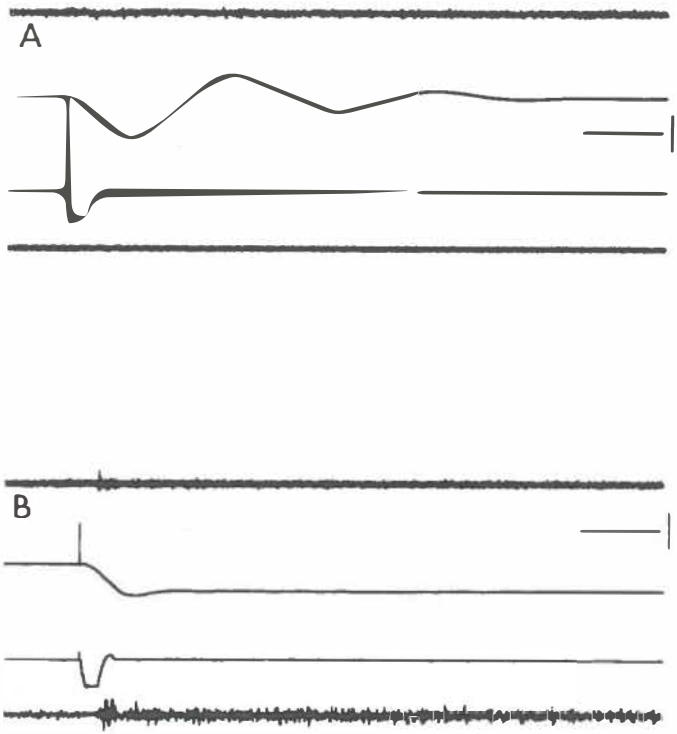

Figure 4.6.:

Plot on two perturbations imposed on the arms of two different subjects. This plot is meant to illustrate the differences between the so-called elastic and plastic behaviour: a. the first subject (HN) showed almost no EMG changes in response to the perturbation: the trajectory of the arm displayed a damped oscillation at the end of which the arm came to a rest at nearly the same position as before the perturbation; b. the second subject (MB) responded to the sudden flexion of the arm with some early stretch activity in the m. triceps, followed by a phasic and tonic shortening reaction in the m. biceps. The increase in EMG activity of the shortening muscle allowed the arm to become stabilized at a new position shifted to a more flexed position than before. Display and calibration as described for figure 4.3.

behaviour: the arm displayed a low damped oscillation. The frequency of this oscillation was about 0.59 Hz[4]. After this oscillation, the arm always settled at nearly the same posture as it

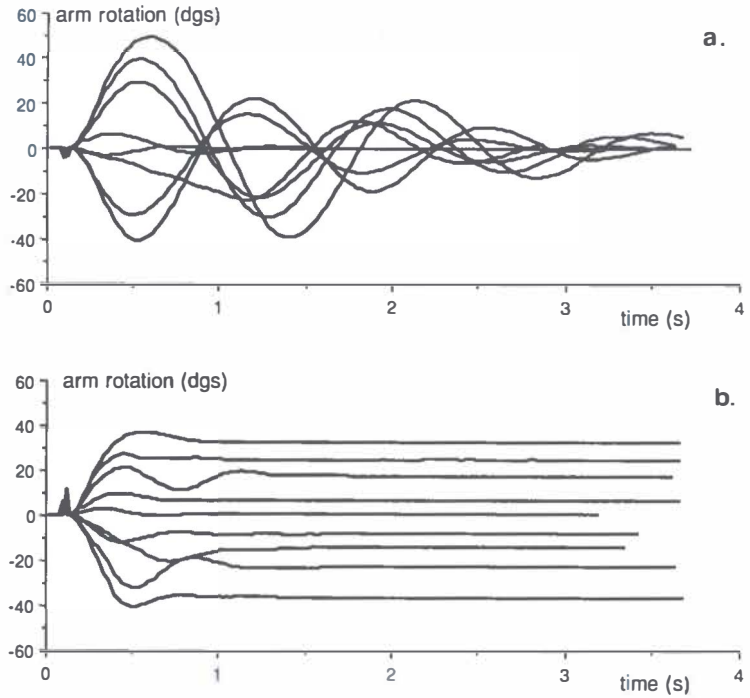

Figure 4.7.:

Plots of the arm trajectories in response to a series of perturbations for the same two subjects as in figure 4.5: a. a subject with elastic behaviour (HN); b. a subject with plastic behaviour (MB). This plot shows that a subject's behaviour remained consistent for a series of perturbations.

[4] It is interesting to deduce from this resonance frequency what is the apparent stiffness of the arm, using the formula $f = 1/(2\pi) \cdot \sqrt{(k/I)}$, with: f designating the resonance frequency, k designating the apparent stiffness and I the moment of inertia of the forearm. For this subject a stiffness was derived of 1.39 Nm/rad (ignoring any contribution of muscle or joint viscosity). This elastic stiffness was ten times less than the median stiffness calculated in the previous paper for the first 100 ms of a perturbation. We take this as evidence that thixotropy reported for relaxed muscles at the wrist (Lakie et al., 1979) also occurs for muscles of the elbow joint.

was in prior to the perturbation. Figure 4.7b. shows the observed arm trajectories after perturbations imposed on the 'plastic type'. This subject gave in to a perturbation and started to conform to no matter what joint angle he was brought to.
The observations made on the subject of figure 4.4., proved to be general for nine of the ten subjects studied; for a certain interval of perturbation amplitudes the arm returned with reasonable accuracy to its starting position, whereas amplitudes outside this interval caused the arm to assume a new position, shifted towards the direction of perturbation.
However, as already mentioned, despite these similarities in behaviour, a considerable amount of inter-subject variability was observed. This is illustrated by figure 4.8., which displays the behaviour of four of our subjects. One subject displayed elastic behaviour: his arms returned to its original positions over a wide range of perturbation amplitudes (Fig. 4.8a.). Another subject displayed plastic behaviour: the arms of this subject conformed to nearly the complete amplitude of a perturbation imposed (Fig.4.8d.). The remaining eight subjects fell on a graded scale between these two extremes (the behaviour of two of these subjects was illustrated in Figs. 4.8b. and 4.8c.).
A plasticity ratio was calculated for each perturbation separately. Subsequently, the median value was taken for the whole series of perturbations imposed on a subject. This median plasticity ratio characterized the behaviour of each subject separately. In this way we obtained a median plasticity ratio of more than 75% for one of our subjects (who was most plastic) and of less than 25% for two other subjects (who were most elastic). The ratios calculated for the rest of the subjects fell in between these two limits (Table 4.1.).

It has already been stressed that little EMG activity was required to maintain the elbow at a given joint angle. Tonic EMG activity was usually less than a few tens of microVolts peak to peak. Though no systematic investigation was undertaken, in general those subjects who displayed plastic behaviour maintained a higher tonic level of EMG activity than those subjects who were of the elastic type. EMG activity of the latter group of subjects would even completely silence once the subjects were fully relaxed. Those subjects displaying plastic behaviour, on the contrary, usually maintained higher levels of tonic EMG activity and, after a perturbation, responded with more obvious lengthening and/or shortening reactions.
The impression that plasticity correlated with a higher level of tonic muscle activation was further substantiated by the finding that a positive correlation existed between the median plasticity ratio and the vividness of the subjects' tendon reflexes (Fig. 4.9.). Scores on

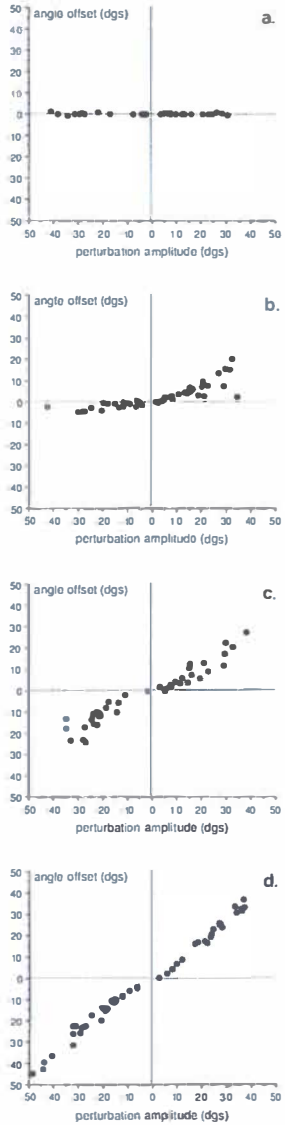

Figure 4.8.:

Plots of the change in maintained joint angle versus maximum perturbation amplitude of all imposed perturbations for four different subjects. The plots were ordered from most elastic to plastic: a. Subject: HN, median plasticity ratio: 0.6 %; b. Subject: RD, median plasticity ratio: 16.9 %; c. Subject: FK, median plasticity ratio: 48.8 %; d. Subject MB, median plasticity ratio: 81.8 %.

the biceps, brachialis, triceps, Mayer and Hoffman-Trömner reflex were added to obtain one figure representing the ease with which these reflexes could be evoked. A statistically significant and positive correlation existed between this score and the obtained median plasticity ratio (Spearman rankcorrelation test, two-sided, $\alpha < 0.05$). Those subjects who displayed more

Subject:	median plasticity ratio: (%)	summed reflex score: (+)
FK	48.8	8
MeB	36.8	9
IN	71.6	14
JeB	63.6	14
MaB	81.8	10
RD	16.9	3
AS	39.2	9
JaB	66.3	14
HN	0.6	8
SC	47.1	7

Table 4.1.:

Table showing the data on median plasticity ratio and summed reflex score for all ten subjects studied. The correlation between these two parameters is shown in figure 4.8.

vivid spinal reflexes were more likely to behave plastically during the perturbation study, whereas those subjects who displayed rather low reflex activity were likely to behave elastically.

4.4. DISCUSSION

The present paper provides evidence that (1) passive movements (or perturbations) of the human forearm induce permanent changes in maintained elbow joint angle. It suggests that these changes in joint posture (2) result from changes in antagonistic muscle activity which are (3) induced by automatic responses within the central nervous system.

Passive movements induce permanent changes in the angle of the elbow joint

By studying permanent[5] changes in joint angle after imposing a passive movement to the forearm it was, in fact, tested how rigidly the elbow joint posture is maintained. The results obtained in this study show that, within the experimental situation, the forearm of a subject returned accurately to its starting position after being perturbed with a small amplitude,

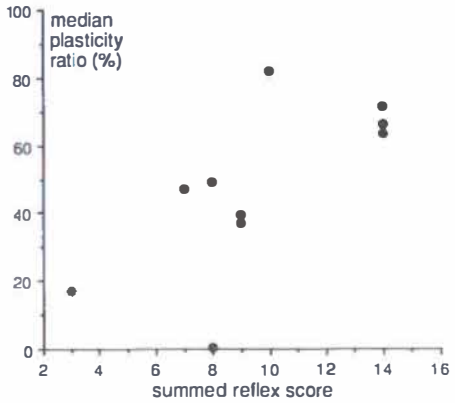

Figure 4.9.:

Plot of the median plasticity ratio versus summed reflex score for all ten subjects (Table 4.1.). This plot shows a statistically significant positive correlation between the vividness of the subjects' tendon reflexes and the amount of postural plasticity observed (Spearman rankcorrelation test, two-sided, $\alpha < 0.05$). This correlation seems contra-intuitive and shows that tendon reflex activity cannot serve as indicator for the degree of rigidity with which posture is maintained.

whereas it conformed to a new position when perturbations were of larger amplitude. This general behaviour is symbolized by figure 4.10.

Nevertheless, a marked variability was found amongst the ten subjects with respect to the range of perturbation amplitudes for which the arm returned to its starting position; some subjects displayed plastic behaviour, since their arm gave in to movements imposed and failed to return to the initial position. Others were elastic, as their arm returned to its starting position over a

[5] 'Permanent' in the sense that the arm, under our experimental conditions, did not return to its starting position unless, as part of the experimental paradigm, it was moved back passively.

wide range of perturbation amplitudes.

For each subject, so-called plasticity ratios were calculated by taking the ratio of angle offset (change in maintained elbow joint angle) and maximum perturbation amplitude. Subsequently,

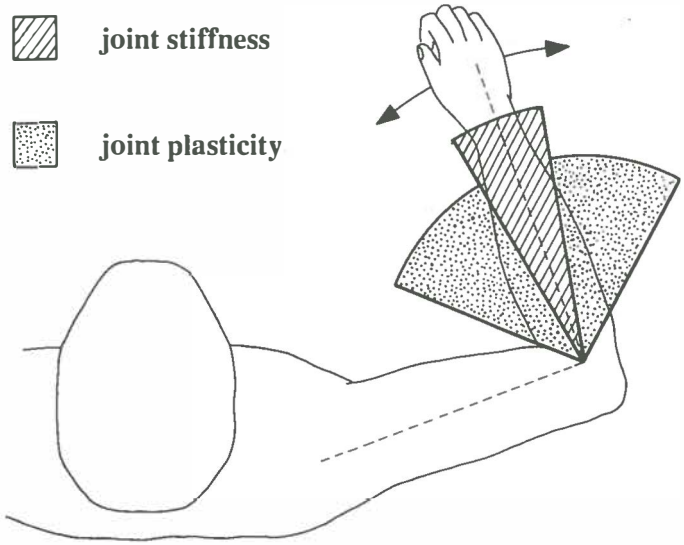

Figure 4.10.:

Schematic representation of the general behaviour of posture maintenance at the elbow joint in response to imposed perturbations. For small perturbations the arm of a subject will return to the original position as result of joint stiffness (or, visco-elasticity) and, supposedly, of the unaltered intrafusal stiction within the antagonists' muscle spindles. For larger perturbations plastic responses occur, possibly intrafusal stiction yields and the limb gives in to the imposed movement: the limb automatically shifts its equilibrium position following the direction of the imposed movement.

the median value was determined of these ratios: for the ten subjects, this median plasticity ratio ranged from 0.6 to 81.8 % (Table 4.1.).

Changes in joint posture are caused by changes in antagonistic muscle activation

From theoretical considerations forwarded by Bizzi and co-workers (Bizzi, Accornero, Chapple and Hogan, 1982a; Bizzi, Chapple and Hogan, 1982b; Bizzi and Abend, 1983) it is deduced

that a relation exists between the differences in activation of two antagonistic muscles and the so-called equilibrium position of a joint.

The force output of a muscle is dependent on its activation as well as on its length: the higher the level of muscle activation, the smaller the length for which the muscle exerts a constant torque. A stable joint angle can only exist when torques exerted by antagonistic muscles are in equilibrium. Therefore, a joint's equilibrium position is determined by the intersection of the torque-angle relationships of antagonistic muscle groups. A joint can only change its position if the balance in antagonistic muscle activation changes.

That different joint angles are generated by differences in antagonistic muscle activation was demonstrated by figure 4.2.: the more active the m. triceps, the more the elbow joint is held in an extended posture, the more active the m. biceps, the more it is held in a flexed posture. Theoretically, a shift in elbow joint posture after an imposed perturbation can, therefore, only be obtained after a change in antagonistic co-contraction. And, as demonstrated in the present study, postural plasticity is often observed to coincide with tonic changes in EMG activity of the mm. biceps and triceps.

Figure 4.2. also shows that, whereas a clear relationship emerges between antagonistic EMG activity and elbow joint posture over the full range of angles studied, a more detailed picture is hard to obtain (e.g. for changes in joint angle less than 5 dgs) as it is impeded by limitations in resolution of the EMG technique[6]. This leaves some space to consider an alternative explanation for the observed shifts in maintained elbow posture after imposed perturbations: they may also have arisen from passive muscle properties, as, for instance, thixotropy (Lakie et al., 1984; Hagbarth et al., 1985). Thixotropy is stiffness which depends on the past history of movement. It is attributed to the formation of stable actin-myosin bonds in resting muscle fibres (Hill, 1968; Lakie et al., 1984). Thixotropy can give rise to an hysteresis in muscle force; when a muscle is stretched and brought back to its original length, the force it exerts is less than prior to stretching.

Theoretically, an hysteresis in muscle force generated by thixotropy may well explain the postural plasticity demonstrated in the present paper. Nevertheless, it is believed that thixotropy is not the right explanation for postural plasticity at the human elbow joint. In the first place, thixotropy has so far only been described to occur in resting muscles (Hill, 1968), whereas, in

[6] Moreover, it should be emphasized that a single joint angle can be maintained by a whole set of different combinations in antagonistic co-contraction (Bizzi et al., 1982a and b, 1983). In order to limit the number of possible solutions, subjects were instructed to maintain a given arm posture as relaxed as possible.

the present study, much care was taken that subjects maintained a definite level of electrical activity in both antagonists. The one subject, who (despite removal of the arm support) displayed little EMG activity in the mm. biceps and triceps, behaved elastically instead of plastically (Fig. 4.5a.). In the second place, thixotropy is expected to manifest itself comparably in different subjects, whereas the present study observed a marked inter-subject variability. Finally, this explanation of postural plasticity would leave unexplained the changes in antagonistic EMG activity accompanying shifts in elbow posture.

Thixotropy is not only described for extrafusal fibres. By its effect on primary afferent activity, it is also demonstrated for muscle spindle intrafusal fibres (Kuffler, Hunt and Quilliam, 1951; Hutton, Smith and Eldred, 1973; Durkovic, 1976; Hagbarth et al., 1985; Gregory, Morgan and Proske, 1988; Gregory, Mark, Morgan, Patak, Polus and Proske, 1990). Thixotropy in intrafusal fibres is usually referred to as intrafusal stiction[7]. It is held responsible for: (1) the initial burst in muscle spindle primary afferent response during a ramp and hold stretch (Hunt and Ottoson, 1976; Proske and Stuart, 1985); (2) for the high spindle sensitivity to minimal changes in length (Brown, Goodwin and Matthews, 1969; Hasan and Houk, 1975) and (3) for after-effects in spindle sensitivity after fusimotor activation (Baumann, Emonet-Dénand and Hulliger, 1983) or after a general muscle contraction (Hnik, Kucera and Kidd, 1970; Hutton, et al., 1973; Smith, Hutton and Eldred, 1974; Gregory, Morgan and Proske, 1988).

Baumann et al. (1983) as well as Hunt (1990) emphasize that the amount of stretch required to abolish the after-effects of intrafusal stiction (>10% of the muscle length) is much larger than the percent change in muscle length required to abolish the muscle stiffness arising from extrafusal stiction (0.2%; Hill, 1968). Out of this, it can be calculated that for the m. biceps an arm extension of 1.2 dgs will suffice to abolish extrafusal stiction, whereas an extension of 60 dgs is required to abolish intrafusal stiction[8]. It should be noted that the 10% muscle stretch of

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[7] It should be realized that the term stiction, in fact, designates a hypothetical mechanism to explain for the high sensitivity to small length changes observed in mammalian muscle spindles. It is questionable whether this high sensitivity is indeed caused by fixed actin-myosin bonds. Other mechanisms should also be considered such as inhomogenous intrafusal contraction (Dickson et al., 1989) or low rate cycling of cross-bridges in passive fibers (Baumann and Hulliger, 1991).

[8] As the fibre length of the m. biceps amounts nearly 150 mm (An, Hui, Morrey, Linscheid and Chao, 1981) a stretch of 0.2% of this fibre length would amount 0.3 mm whereas a stretch of 10% would amount 15 mm. As the working arm of the m. biceps at an elbow angle of 80 degrees is about 14 mm (An et al., 1981), the corresponding change in elbow angle for a stretch of 0.3 mm would amount $\sin^{-1}(0.3 / 14) = 1.2$ dgs. A stretch of 15 mm can be seen as

Hunt (1990) serves to abolish all after-effects in spindle sensitivity; it was calculated in our previous paper that the first yielding of intrafusal stiction may occur at an elbow extension of 2 dgs. Again, these calculations indicate that intrafusal stiction may be of greater significance for posture maintenance at the human elbow joint than is its extrafusal counterpart (compare Lakie et al., 1984).

These considerations led us to conclude that a permanent shift in elbow angle occurred because of changes in antagonistic muscle activation and not because of passive muscle properties. We will now briefly discuss the observed changes in EMG activity as well as their possible physiological origins.

It should be noted, that only tonic changes in EMG activity are relevant for the observed shifts in elbow angle. Many phasic responses have been observed during and after an imposed perturbation, both in the EMG of the stretched as well as the shortened muscle. However, all phasic responses are of no effect upon the final elbow joint posture. The final angle of the elbow joint is fully determined by the equilibrium position of Bizzi et al. (1982a; 1982b; 1983) determined by the intersection of both antagonistic torque-angle relationships.

It can at least be said, on basis of the present study, that phasic EMG responses to a perturbation are ambiguous; apart from the often observed short latency burst of EMG activity in the stretched muscle, we often observed an increase in EMG activity of the shortening muscle (previous paper, Fig. 4.2.; present paper, Figs. 4.3b., 4.4a., 4.4d., 4.5a., 4.5b., 4.5c. and 4.6b.) as well as, occasionally, an inhibition of the activity of the stretched muscle (previous paper, Fig. 4.8.; present paper, Figs. 4.5b. and 4.5c.). We have not seen long latency stretch reflexes described by other investigators (Hammond et al., 1956; Nashner, 1976; Marsden et al.,1983), which is probably due to the instruction of our subjects (compare Figs.4.3b. and 4.3c.).

A decrease in muscle activation during muscle stretch, is known as lengthening reaction. It is, for instance, present in some of the recordings of Angel (1983) and of Latash and Gottlieb (1990). An increase in muscle activation upon muscle shortening is known as shortening reaction. Shortening reactions have, under different experimental conditions, been observed by several other investigators (Andrews, Burke and Lance, 1972; Angel, 1983; Gottlieb and Agarwal, 1988; Latash and Gottlieb, 1990).

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two times a stretch of 7 mm, corresponding with 2 x $\sin^{-1}$ (7 / 14) = 60 dgs extension (e.g. from 50 dgs to 110 dgs).

The shortening response is called a 'paradoxical response' (Westphal, 1880), because, in contrast to the well known myotatic reflex, it does not seem to aim at restoring joint posture. On the contrary, the combined action of shortening and lengthening response seems to facilitate a change of posture. They are held responsible for the postural plasticity observed in the present study. Their physiological origin is largely unknown (Angel, 1983). It is suggested that they arise from changes in inhibitory feedback, originating from Ib afferents (Andrews et al., 1972). Muscle stretch, for instance, may induce a rise in Ib-afferent discharge by the activation of Golgi tendon organs, and, thereby, cause inhibition of autogenetic alpha motoneurons. A sudden drop in muscle tension resulting from muscle shortening, in contrast, would cause a dis-inhibition of autogenetic alpha motoneurons, resulting in the shortening reaction.

The present study has made no attempt to elucidate the physiological mechanism by which the shortening and lengthening responses occur. However, it does suggest a functional role of these responses for posture maintenance at the human elbow joint. Whereas myotatic reflexes serve to maintain joint stability, the lengthening and shortening reactions serve to provide postural plasticity. Postural plasticity is just as important as postural rigidity, if the organism is to assume different joint angles instead of a single one.

However, a large inter-subject variability was observed, ranging from elastic to most plastic. Should it be inferred that some subjects do not dispose of postural plasticity? The large variability suggests that, when confronted with our experimental set-up, subjects may chose a different pre-setting of their responses. The two subjects who were most elastic were trained athletes, who had a well developed ability of voluntary muscle activation and relaxation. Though arm support was denied, they were still able to relax their elbow muscles so that they silenced completely. After a perturbation, their arm trajectory displayed a low damped oscillation; its end position was always that of iso-elasticity, no matter with what amplitude or velocity it was perturbed. In contrast, when arm support was denied to other subjects this resulted in a co-contraction of elbow antagonists. These subjects displayed plastic behaviour.

Changes in tonic EMG activity are caused by automatic responses within the CNS

It is our conviction that the observed changes in antagonistic EMG activity, shortening as well as lengthening reaction, are elicited by automatic mechanisms occurring within the CNS in response to an imposed perturbation. This conviction was based on the fact that, during the experiment, the subject's attention could be distracted from his (or her) arm; we started a vivid conversation or allowed the subject to read. Another argument supporting the assumption that

responses were automatic, was that some of the subjects occasionally expressed their surprise that they were unable to tell whether a motion was caused by the torque motor or by their own muscle action. It should be noted that the term 'automatic' is used to designate all nervous responses that occur without necessitating a subject's alert attention.

The latency of the shortening response was always observed to be larger than that of the stretch reflex. An observation consistent with that of Andrews et al. (1972). In the present study we found stretch reflexes with a latency of 30-40 ms, whereas the first signs of a shortening reaction were found at 90-100 ms after perturbation onset. These differences in latency suggest that the postural system, at first, tries to correct for a perturbation, but later gives in and facilitates the imposed movement of the arm.

Conclusion

In our previous paper (Schaafsma and Van Willigen, 1991b) it is shown that posture maintenance at the human elbow joint is achieved by the co-contraction of elbow antagonists which establishes a local minimum in an energy landscape in which the arm lies. It is suggested that the stable localization of this energy minimum is ensured by the activity of myotatic reflexes, of which the action is based upon a high spindle sensitivity possibly arising from intrafusal stiction. Hammond et al. (1956) have already stressed the advantages of a postural system making use of muscle spindle afferent activity to maintain the steady activation of alpha-motoneurons required for a stable joint posture.

We now suggest that differences in plasticity ratio found between small and large imposed perturbations of the forearm, are brought about by the same mechanism. A small perturbation does not disturb the intrafusal stiction established at the starting posture of the elbow, and the forearm will return to its original position; whereas a large perturbation may cause yielding of intrafusal stiction, resulting in a decreased spindle sensitivity, and allowing the forearm to become stabilized at a new and different elbow angle.

Baumann et al. (1983) have demonstrated that after-effects in muscle spindle primary afferent activity may be enhanced during and after fusimotor activity[9]. We suggest that the inter-subject variability correlates with differences in fusimotor tone. When fusimotor tone is high, a subject will display a higher level of antagonistic co-contraction; but also, due to the activation of intrafusal muscle fibres, the spindle will display a greater adaptive capacity to changing muscle

[9] This is in contrast with the short range elasticity component in extrafusal fibres. Hill (1968) shows that during tetanic muscle activation, the short range elasticity (or thixotropy) rapidly disappears, as cross-bridges can no longer remain stable when they are actively cycling.

lengths, resulting in postural plasticity. When fusimotor tone is low, a subject will display a low level of antagonistic co-contraction; the spindle will not so readily follow a change in muscle length and the subject will behave elastically.
Such a tentative explanation of the results of the present study is indirectly supported by the positive correlation found between the subjects' median plasticity ratio and the vividness of their tendon reflexes (Fig. 4.9.). Postural plasticity was observed most in subjects with a high level of antagonistic co-contraction and with vivid tendon reflexes; postural plasticity was observed in subjects with a low level of co-contraction and weak or absent arm reflexes.

Concluding, the present and previous paper suggest that the role of the myotatic reflex is to stabilize the localization of an energy minimum in which the arm lies. Though the synaptic gain of the myotatic reflex is low (Matthews, 1966), this is compensated for by the high spindle sensitivity occurring as result of intrafusal stiction (Matthews, 1981; Schaafsma and Van Willigen, 1991b). Also, intrafusal stiction provides a great adaptive capacity to the postural system, since it allows the high length sensitivity of muscle spindles to establish at any given muscle length (Matthews, 1972; Hulliger 1981 and 1984; Gregory et al., 1990). Thereby, the contribution of the myotatic reflex is one of great subtlety: the high gain with which the stretch reflex controls a limb posture, disappears instantaneously as soon as a (passive or active) limb movement occurs, since the movement itself causes the intrafusal stiction to yield.
When viewed in this way, the physiological function of myotatic reflexes may easily escape from our experimental attention as it generates only slight changes in EMG activity in response to minute changes in joint angle over prolonged periods of time. The synchronized burst of EMG activity evoked by a tap with a tendon hammer should then be regarded as an oversized manifestation of this subtle control mechanism; it only proves the integrity of pathways required for its functioning. Therefore, it comes as no surprise that monosynaptic stretch reflexes contribute little to the mechanical responses aimed at restoration of posture after a large perturbation (Hammond et al., 1956; Marsden et al., 1983). Large perturbations can only be corrected by the mechanical effects of late components in EMG activity (automatic as well as voluntary; Nashner, 1976).

CHAPTER 5

A modelling approach

5.1. INTRODUCTION

The aim of this chapter is to describe a model simulating the results of experiments on posture maintenance at the human elbow joint (Schaafsma and Van Willigen, 1991b/c). In this introduction, we will briefly review what the results of this study were.

The study of Schaafsma and Van Willigen (1991b/c) deals with the (automatic) control of elbow joint posture. Passive movements (perturbations), randomized with respect to amplitude and velocity, are imposed on the forearms of volunteers by means of a torque motor. The subjects are instructed not to react against these movements. Under these conditions, the arm of each subject displays a characteristic behaviour, which is thought to arise from involuntary responses of the subject to the imposed movements of the arm.

In their first paper, Schaafsma and Van Willigen (1991b) plot the amount of work done by the torque motor in order to rotate the arm over a certain distance. This plot illustrates the amount of energy required to rotate the arm away from its resting angle. This plot is also an illustration of how the unperturbed arm lies stabilized in a local energy minimum. It is argued that the steepness of the slopes of this energy minimum is dependent upon the amount of antagonistic co-contraction.

In their second paper, Schaafsma and Van Willigen (1991c) compare the starting position of the arm before a perturbation with that at which the arm becomes stabilized after a perturbation. They show that, for small perturbation amplitudes, the arm returns accurately to its original position. For larger perturbation amplitudes, however, it becomes stabilized at a new position, shifted in the direction of the perturbation.

A return of the arm to its original position may be expected either when the activity of the elbow muscles remains unaltered (postural elasticity) or when muscle activity is changed on basis of stretch reflex activity (postural rigidity). Prolonged changes in elbow joint posture are shown to

be accompanied by tonic changes in antagonistic muscle activation (postural plasticity). These changes in muscle activation are exactly opposite to those expected on basis of the myotatic stretch reflex: the shortening muscle increases its EMG-activity, whereas the lengthening muscle decreases its activity.

So, a characteristic behaviour can be observed in response to perturbations of variable amplitude: small perturbations are effectively overcome by posture restoring mechanisms, whereas large perturbations elicit involuntary responses which allow the arm to change to a new joint position. Consequently, posture maintenance at the human elbow joint is a mixture of mechanisms opposing and mechanisms supporting a passive movement of the arm.

Based on the observations described we suggest that posture maintenance at the human elbow joint is achieved by a balanced tuning by the central nervous system (CNS) of elbow extensor and flexor torques. The difference in antagonistic muscle activation determines the angle of the joint (Bizzi and Abend, 1983), and the parity in activation the stiffness of the joint (Humphrey and Reed, 1983). Moreover, we suggest that posture is maintained by the constancy of antagonistic muscle activation brought about by the negative length feedback originating from the action of the myotatic reflex. Muscle spindles display a remarkably high sensitivity to changes in muscle length, which allow them to respond promptly to any drift in joint posture. However, the high length sensitivity of muscle spindles is of a limited range, since a change of spindle length of a few tens of micrometers causes the high sensitivity to disappear (Hasan and Houk, 1975). We hypothesize that under these circumstances the myotatic reflex is no longer able to bring the arm back to its original position and plasticity responses take over. These responses cause a shift in position of the set-point of maintained joint posture.

This chapter aims to investigate whether this theory about posture maintenance at the human elbow joint proves valid when implemented in a computer simulation. It should be emphasized that this simulation is no more than an illustration of this theory in which there are still many uncertainties.

The model is partly an integration of models developed earlier at our laboratory (Otten, 1987b; Schaafsma et al., 1991a). It consists of: (1) a model of the human elbow joint with muscle lever arms depending on the angle of the joint; (2) a dynamic muscle model, predicting force output on the basis of muscle activation, architecture, length and contraction velocity; (3) a muscle spindle model predicting Ia and II-afferent activity out of muscle spindle length, stretch velocity and fusimotor conditions; (4) a model of presumed reflex mechanisms, simulating proprioceptive information processed by the central nervous system.

5.2. METHODS

The model of posture maintenance at the elbow joint was constructed out of four submodels. Each submodel will be described separately.

A model of the forearm and the elbow joint.
Schaafsma and Van Willigen (1991 b/c) use an experimental set-up which only allows the subject's arm to move in a horizontal plane. Therefore, forces of gravity can be ignored since they do not result in any flexing or extending torque over the joint.

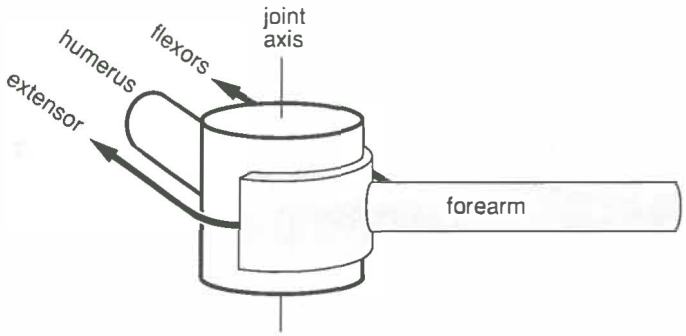

Figure 5.1.:

A schematic representation of the elbow joint used as basis for the model of the arm. The elbow joint is represented as an ideal cylindrical joint with its axis perpendicular to the horizontal plane. In this way any rotation around the joint axis will not change the forearm's potential energy. The joint angle is determined by the equality in torque exerted by elbow extensor, the m. triceps, versus elbow flexors, the mm. biceps and brachialis.

In the computer simulation, a simple cylindrical joint was taken as model of the elbow joint: we assumed that the rotational axis of was perfectly perpendicular to the horizontal plane (Fig. 5.1.). The forearm was modeled as an inertial element rotating around the axis of the cylindrical joint. Its inertia was (arbitrarily) set at 0.09 $kg \cdot m^2$. This figure is based upon measurements, at our laboratory, of forearm moment of inertia (ranging from 0.043 to 0.115 $kg \cdot m^2$) in fifteen

subjects, including the ten subjects in the study of Schaafsma and VanWilligen (1991b/c). Data were obtained as described in these papers.

A model of the muscles governing the elbow joint.

As gravity is prevented from exerting torque over the elbow joint, the angle of this joint depends only on the force output of the muscles crossing it. These are: the m. triceps and m. anconeus on the extensor side and the m. biceps, m. brachialis and m. brachio-radialis on the flexor side. The m. anconeus and the m. brachio-radialis have been excluded from this study; the m. anconeus since it is relatively weak as estimated from its physiological cross sectional

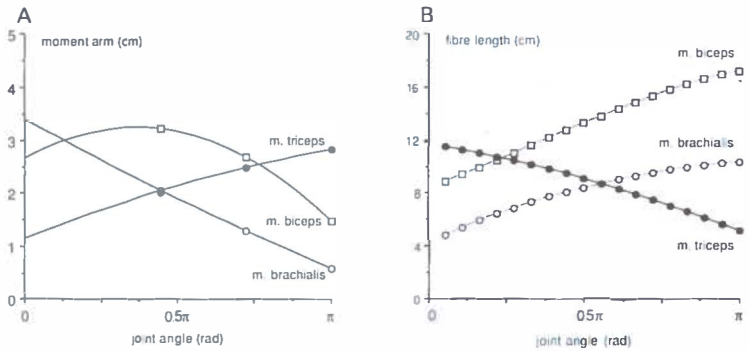

Figure 5.2:

A. Data on the relationship between moment working arms of the elbow extensor and flexors (dots and squares) according to An et al. (1981). These data have been fitted by a second order polynomial (continuous line).
B. Plot of muscle fibre length as function of elbow joint angle as reconstructed from the data on moment working arms of An et al. (see text).

area when compared with the triceps (An, Hui, Morrey, Linscheid and Chao, 1981), the m. brachio-radialis because experiments at our laboratory have pointed out that its EMG activity changes minimally with elbow angle.

Muscle forces were modeled as single force vectors acting on the forearm. It was assumed that the direction of these force vectors was always in parallel with the axis of the model's humerus (Fig. 5.1.). The lever arms over which the muscle force vectors acted were derived from a study of An et al. (1981). We did not take into account muscle compartmentalization: for each

muscle only one (average) lever arm was calculated. In this way, we obtained lever arms at three different elbow angles which we fitted with a polynomial of the second order (Fig. 5.2a.). By making use of measurements on the muscle fibre length at 110 degrees elbow flexion (An et al., 1981), we were able to describe the relationship between muscle fibre length and joint angle as a third order polynomial function (Fig. 5.2b.). This was achieved by integration of the second order relationship between lever arm and joint angle (Fig. 5.3.).

	m. biceps:	**m. brachialis:**	**m. triceps:**
a	-0.4445	0.0394	0.0657
b	1.0230	-1.0153	-0.7391
d	2.6412	3.3928	-1.1350
c	8.3924	4.2645	11.6863

Table 5.1.:

Parameter values of a,b,c and d for each modeled muscle, determining the relationship between lever arm and joint angle (Fig. 5.2a.) and muscle fibre length and elbow joint angle (Fig. 5.2b.).

The data of An et al. (1981) consisted of measurements on rotational lever arms for various muscles, at three different angles of the elbow joint, namely at 80, 130 and 180 degrees extension[1]. A polynomial fit of the second order was calculated through these data:

(1) $R_i(\alpha) = a_i \cdot \alpha^2 + b_i \cdot \alpha + c_i$

R denotes the lever arm of the muscle i, α the joint angle in radians and with the values of a, b and c provided by Table 5.1.
Figure 5.3. shows that, by approximation, the change in muscle fibre length is the lever arm times the change in joint angle. Therefore:

(2) $\Delta L_i = R_i(\alpha) \cdot \Delta \alpha$

[1]Some confusion might arise as An et al. (1981) have chosen a different co-ordinate system with 0 degrees referring to maximal extension and 180 degrees to maximal flexion. We preferred the mirror-image of this system: maximal flexion is referred to as 0 degrees and maximal extension as 180 degrees.

L_i denotes the muscle fibre length of muscle i. In order to calculate the muscle fibre length out of the elbow joint angle we integrated (2) and obtained:

(3) $L_i(\alpha) = \int (R_i(\alpha) \cdot d\alpha)\, d\alpha$

$L_i(\alpha)$ denotes the muscle fibre length of muscle i at the elbow angle α in radians. Out of (1) it follows that:

(4) $L_i(\alpha) = a_i \cdot 1/3 \cdot \alpha^3 + b_i \cdot 1/2 \cdot \alpha^2 + c_i \cdot \alpha + d_i$

$L_i(\alpha)$ denotes the muscle fibre length of the muscle i at the joint angle α in radians and with the values of a, b, c and d according to Table 5.1. The constant d could be derived from data of An et al. on the muscle fibre length of each muscle for a joint angle of 110 degrees extension.

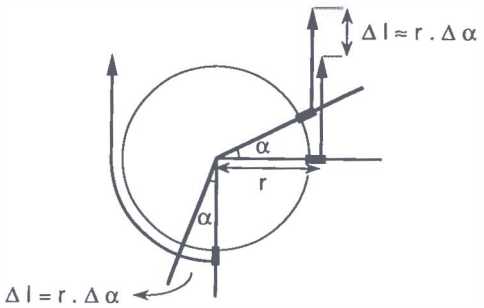

Figure 5.3.:

This figure shows how a change in muscle fibre length will depend on a change in joint angle when the lever arm (r) is considered to be constant. Any change in muscle fibre length (Δl) will, in approximation, be a function of the lever arm times the change in joint angle (Δα) expressed in radians. In the model calculation the level arms were not taken constant but depended on the joint angle α (compare fig. 5.2a.).

In order to calculate muscle forces, we used the dynamic muscle model formulated by Otten (1987b). This model is based on a static muscle model (according to Otten, 1987a: Fig. 5.4a.) and on a dynamic extension of this model (in agreement with data of Hill, 1938 and Aubert , 1956: Fig. 5.4b.). The static part of the muscle model calculates an output force by adding a passive muscle force (as function of muscle length) with an active muscle force (as function of

muscle length and muscle activation). The dynamic part of the muscle model scales the active muscle force by multiplication with a factor depending on the muscle contraction velocity (force-velocity curve of Fig. 5.4b.).

Basically, the dynamic muscle model of Otten, provided that its parameters are set, allows the calculation of muscle force for any given combination of muscle length, muscle activation and muscle velocity (Fig. 5.5.). The parameter-values which we used in the model of the human elbow joint are given by Table 5.2.

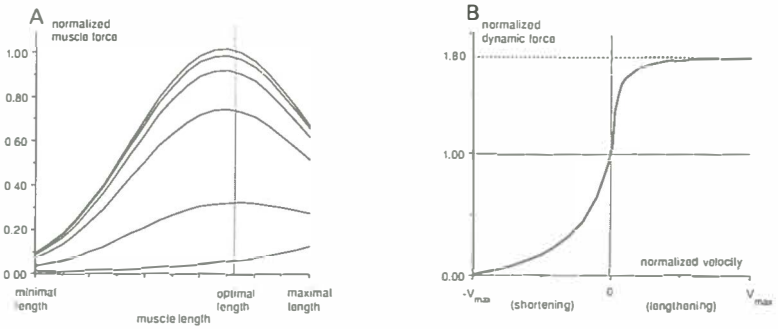

Figure 5.4:

A. Graph illustrating the relation between muscle force and muscle fibre length (Otten, 1987b) implemented in the model of the arm. The bottom most trace shows the passive force-length relationship. The remaining five traces show, from bottom to top, the simulated force output at 20, 40, 60, 80 and 100% of maximal muscle activation. The optimal muscle fibre length (dotted line) was chosen at 2/3 of the physiological range (Alexander, 1989). Maximal force output is attained at a length slightly shifted to the left as passive muscle force needs to be taken into account also.
B. Graph illustrating the relation between muscle force and muscle contraction velocity implemented in the model of the arm. This figure shows by what factor the active muscle force is multiplied in order to obtain a dynamic muscle force. The lengthening part of the force velocity curve was taken according to Otten (1987b), the shortening part according to Hill (1938). The maximal shortening velocity was based on measurements of Close (1964; see Table 5.2.).

Firstly, a maximum force output was determined for all three muscles of the model. These were derived from data of Singh and Karpovich (1965) on the maximal isometric flexion and extension force measured at an elbow angle of 90 degrees flexion. They measured a maximum force output of 45.40 lb = 201.8 N for flexion and of 29.43 lb = 130.7 N for extension. Singh and Karpovich were unclear about the lever arm at which they measured these forces. We have

	m. biceps:	m. brachialis:	m. triceps:	references:
maximal force output	1110.3 N	1689.5 N	2015.0 N	(Singh and Karpovich 1965)
	----------	all three muscles:	----------	
optimal fibre length	at 2/3 of the physiological range			(Alexander, 1989)
V_{max}	13.7 times the optimal fibre length			(mean of 7.1 and 18.3; Close 1964)
Hill constant k	0.21			(mean of 0.17 and 0.25; Close 1964)
$[Ca^{2+}]_{max}$	2 μM			(Otten 1987b: Julian and Moss 1981)
$[Ca^{2+}]_{0.5}$	0.59 μM			(Otten 1987b: Julian and Moss 1981)
c_q	60			(Otten 1987b: Wallinga-De Jonge 1980)
c_r	33			(mean of 16 and 50; Otten 1987b:Wallinga-De Jonge 1980)
roundedness a	2.1			(Otten 1987b)
skewness b	1.0			(Otten 1987b)
width s	0.3			(Otten 1987b)
c_1	-7			(arbitrary)
c_2	4			(arbitrary)
F_c	0.3			(Otten 1987b)

Table 5.2.:

Parameter settings for the dynamic muscle model. The same notations have been used as in the original paper of Otten (1987b). For several parameters the values were calculated by averaging the values for slow and fast muscle fibres, respectively, on the assumption that the distribution of fast and slow muscle fibres within each of the three muscles would be equal and would be half fast and half slow.

taken 0.33 m as the length of the forearm and calculated a maximal muscle torque of 67 Nm for the flexors and 43 Nm for the extensors. As the flexion torque depended both on the m. biceps and on the m. brachialis, it was necessary to subdivide the total flexion torque into the two torques of either muscle. For this we used figures of An et al. (1981) on the physiological cross sectional area of the two muscles. The cross sectional area of the m. brachialis is 1.52 times larger than that of the m. biceps. The force output of both muscles should relate equally. However, the m. biceps (at 90 degrees flexion) acts over a lever arm of 0.0315m, whereas the lever arm of the m. brachialis acts over an arm of 0.019m. Taking this into account we calculated a maximal isometric force for the m. biceps of 1110.3 N, for the m. brachialis of 1689.5 N and for the m. triceps (with a lever arm of 0.0213m) of 2015.0 N.

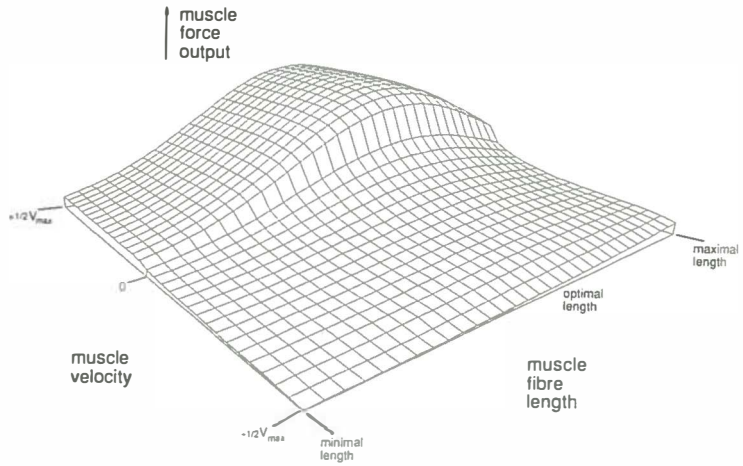

Figure 5.5.:

A three dimensional plot of the model's muscle force output for different combinations of muscle length and muscle velocity at a constant muscle activation of 50% of maximal.

Furthermore, we defined a scaling of the model's muscle fibre lengths to the normalized lengths of the dynamic muscle model. This boils down to deciding on an elbow angle at which optimal sarcomere length is achieved (producing maximal active force output) and how this sarcomere length changes with muscle fibre length.

The optimal sarcomere length was chosen to be reached at 2/3rd of the physiological range of each muscle (Alexander, 1989; see Fig. 5.4a.). The physiological range of a muscle refers to the range of muscle fibre lengths obtained between maximal flexion and maximal extension of the elbow joint. In addition, appropriate values were chosen for the skewness, the roundedness and the width of the active curve (a, b and s in Table 5.2.), and for the values of c_1, c_2 and F_c of the passive curve. Thereby, we further scaled the model's force output with respect to the muscle fibre length.
The remaining parameters were set according to Otten (1987b). Some of these depended on the ratio between fast and slow muscle fibres. This ratio was chosen fifty-fifty for all muscles within the model. The values for the parameters depending on this ratio have, therefore, been obtained by averaging literature data on fast and slow muscle fibres, respectively.
Finally, an extra viscosity was introduced within the model, in order to account for joint viscosity. This was mainly done for reasons of stability. The value chosen for the joint viscosity amounted 0.1 Ns/m and was, thereby, within the range provided by MacKay et al. (0.08 to 0.3 Ns/m; 1986). It should be noted that the data of MacKay et al. were obtained by optimization of free parameters within a model of their set-up in order to simulate experimentally derived data.

Now that all parameters of the dynamic muscle had been set, we were able to calculate the relationship between muscle torque and elbow joint angle for different levels of muscle activation. This is illustrated in figure 5.6., which displays muscle torques for activations of 0, 10, 20, 30, 40 and 50% of maximum. The negative torques exerted by the combined action of the mm. biceps and brachialis have been inverted in order to facilitate their comparison with the positive torques exerted by the m. triceps. Though the force-length relationship (Fig. 5.4a.) of each muscle was asymmetrical, the torque angle-relationship was bell-shaped. Apparently, the steep rise in muscle force seen with increasing muscle length was not seen in the net muscle torque due to the decrease in muscle lever arm (Fig. 5.4a.).
Intersections of the flexor and extensor curves of figure 5.6. represent so-called equilibrium points (Bizzi and Abend, 1983). These equilibrium points need not all be 'stable'. Equilibrium points are considered stable when small changes in arm position are antagonized by the resulting changes in muscle torque. For instance, if we consider the intersection of the 50% activated extensor curve and the 20 % activated flexor curve (indicated by the asterisk in Fig. 5.6.) a small drift of the forearm towards extension will cause both a drop in extensor as well

as in flexor torque. However, the extensor torque decreases more rapidly than the flexor torque, and the net torque will direct the arm back to its starting position: this equilibrium point is a stable one. If, instead, the flexor torque would have decreased more rapidly than the extensor torque, this position would be unstable, and the net torque would have directed the forearm further away from its starting position.

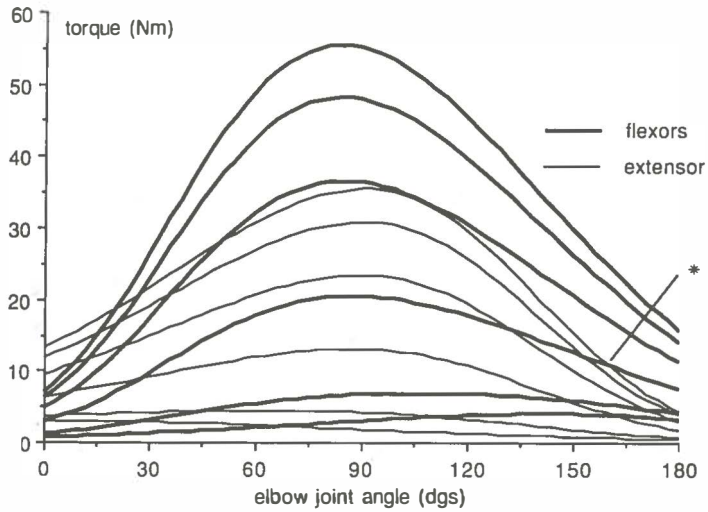

Figure 5.6.:

Torque-angle relationship for the modeled flexor and extensor muscles under 0, 10, 20, 30, 40 and 50% of maximal muscle activation. Each inter-section of a flexor and extensor curve displays a stable equilibrium point; for this particular combination of flexor and extensor activation the arm can rest stable at the corresponding joint angle.

In other words, intersections within the torque-angle plot are stable when, for a small extending movement of the forearm, the net torque becomes negative and are unstable when the net force becomes positive.

A model of muscle receptor activity.

Schaafsma et al. (1991a) formulate a model for muscle spindle primary afferent firing based on a simulation of intrafusal mechanical events. It consists of two models of nuclear bag fibres: one simulating the mechanics of the dynamic bag_1, the other of the static bag_2 fibre. Fibre

mechanics were based on the dynamic muscle model of Otten (1987b). The muscle spindle model produces afferent discharges which nicely resemble responses observed from real muscle spindles.

For the implementation of the muscle spindle model within the flexor and extensor muscle models we defined how a change in muscle length would relate to a change in spindle length. Under passive (de-efferented) conditions the muscle spindle model may be stretched up to 10 mm before it reaches its firing-threshold. It was chosen to let this 10 mm spindle length correspond with a muscle fibre length at 30% of the physiological range. This choice also determined what proportion of a change in muscle fibre length would be transmitted to the muscle spindle.

The muscle spindle model allows its sensitivity to be adjusted by varying the dynamic and static fusimotor conditions as in real spindles. However, for the purpose of the present study it was chosen to keep fusimotor conditions constant: dynamic fusimotor activity was switched off in order to obtain a maximal effect of intrafusal stiction (see hereafter); static fusimotor bias was chosen at 20% of maximal[2]. Under these fusimotor conditions, the spindle discharge at different muscle fibre lengths is illustrated by the heavy line in figure 5.7. As can be seen, the static spindle discharge increases exponentially as a function of muscle fibre length[3]. The thin lines in figure 5.7. show the dynamic discharge of the spindle model in response to muscle stretch and release at two different velocities. Starting off from its static discharge, the spindle

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[2] In this first modeling attempt, these values were not chosen in agreement with what is known from real muscle spindles. Instead, the possibilities and limitations of the implemented muscle spindle model served as main indicator for the choice of fusimotor conditions. Dynamic fusimotor activity was chosen zero, in order to have a maximum effect of stiction in the modeled dynamic bag1 fibre. Static fusimotor activity was chosen at 20% of maximal in order to (slightly) increase the model's length sensitivity. It should be realized, that in the model, the static and dynamic innervation serve two strictly separated compartments. Therefore, static fusimotor activity will not, as in real muscle spindles, interfere with the mechanism of intrafusal stiction.

[3] The exponentional increase in static Ia-activity is caused by the passive muscle elasticity incorporated in the modeled intrafusal fibres. Literature studies on the primary afferent discharge when the spindle is stretched over its full physiological range do not agee with such a relationship (Hulliger et al., 1982). It is not overtly clear how this dispcrepancy arises. One possibility is that intrafusal nuclear chain fibres have a more linear length-tension relationship as compared with extrafusal fibres. Nuclear chain fibres contain an increased quantity of elastin fibres and because of this display so-called kinking on release (Boyd, 1976). It is not inconceivable that the parameters used for modeling the static bag_2 fibre (which in the model also represents the activity of the nuclear chain fibres) should be modified when more information comes at hand about the kinking of nuclear chain fibres.

model produces a steep increase in activity in response to muscle stretch.

The muscle spindle model of Schaafsma et al. (1991a) contained a simulation of so-called intrafusal stiction. This is the phenomenon that particular sarcomere groups of the dynamic bag_1 fibre possess cross-bridges (actin-myosin bonds) which are stable and not cycling[4]. These

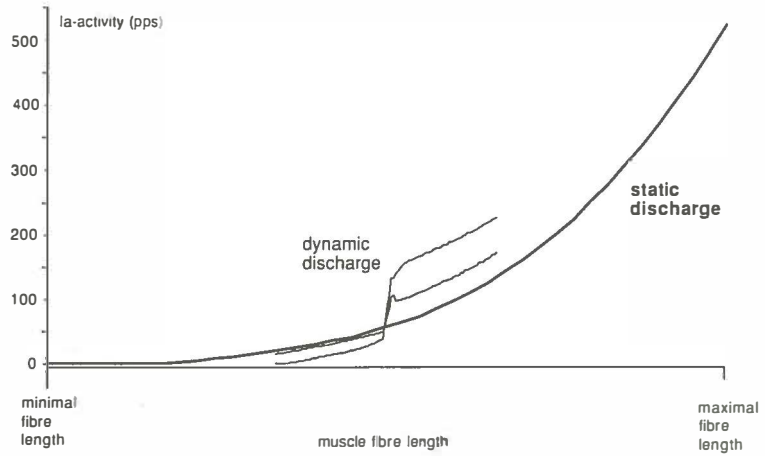

Figure 5.7.:

Implementation of the muscle spindle model of Schaafsma et al. (1991) within the modeled arm muscles. The spindle model was held under constant fusimotor conditions of 0 Hz dynamic and of 20 Hz static gamma activity.
Heavy line: static discharge of the spindle model; thin line: its dynamic discharge. Dynamic discharges are obtained at stretch (or release) velocities of 0.2 and of 1 times the optimal muscle fibre length/second.

fixed sarcomere groups cause the bag_1 fibre to display a short range elasticity comparable to that observed in extrafusal fibres (Hill, 1968). When the intrafusal fibre is stretched, stiction will cause most of this stretch to be transmitted directly to the sensory region. Stiction, therefore,

[4] At least, this is one hypothesis explaining how the muscle spindle can display such extreme sensitivity to small changes in length. It has been demonstrated that the spindle's high length sensitivity derives from mechanical factors (Hunt and Ottoson, 1976). Whether this indeed is due to stable actin-myosing bonds, and not to other intrafusal mechanisms such as, for instance, an atypical form of contraction in the nuclear bag_1 fibre, remains to be proved. We have used the term stiction in a somewhat looser sense to express whatever mechanical stickiness may underly the spindle's high sensitivity to small changes in length.

enhances the muscle spindle's length sensitivity. As in extrafusal fibres, stiction within the muscle spindle has a limited range. Hulliger et al. (1977) have shown that muscle spindles in

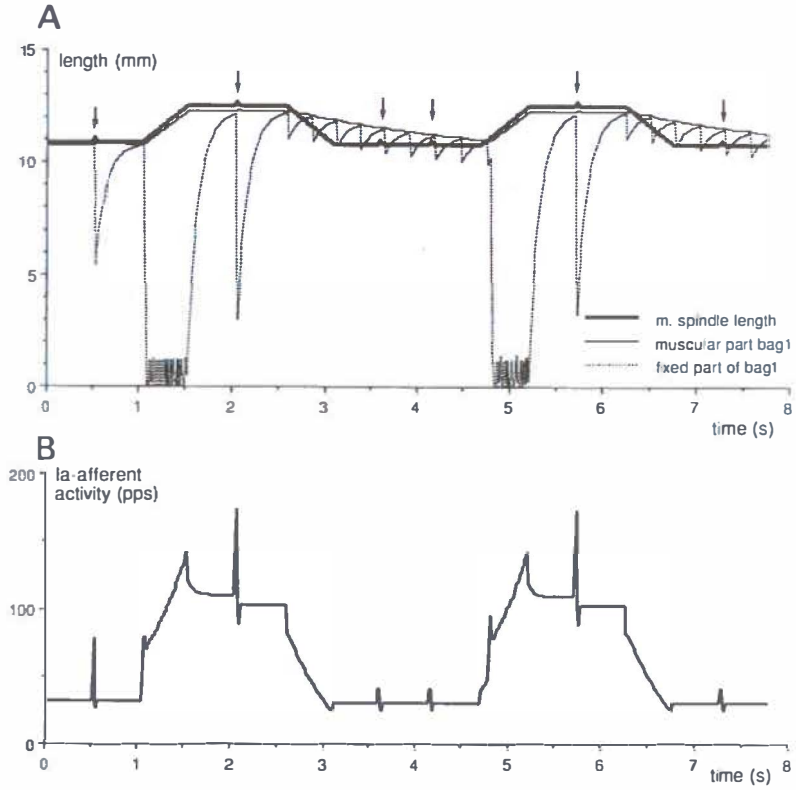

Figure 5.8.:

This figure illustrates the effect of 'intermittent stiction' with which the muscle spindle model of Schaafsma et al. (1990a) was expanded.
A. The fat line displays the overall length of the muscle spindle model. The continuous line displays the part of the overall spindle length taken up by the muscular part of the dynamic bag_1-fibre. Depending on previous streches or releases, part of the bag_1 muscular part may become 'free' or 'fixed'. The dotted line represents the extent to which the muscular part of the bag_1-fibre is fixed.
B. Based on intrafusal mechanical events the muscle spindle model calculates a Ia-afferent discharge (fat line). Several small test stretches (arrows) show what effect yielding and re-attachment of crossbridges within the dynamic bag_1-fibre have on the overall spindle sensitivity. Large stretches and releases prove capable of temporarily changing the spindle's sensitivity. (For further explanation see text.)

the cat soleus display a high sensitivity to sinusoidal stretching as long as the amplitude of stretching remains smaller than 100 μm (amplitudes larger than 100 μm are assumed to cause yielding of intrafusal stiction and, thereby, a decrease in muscle spindle length sensitivity).

In the spindle model of Schaafsma et al. (1991a) stiction does not re-establish once it has yielded. For the present model, however, it proved necessary to introduce 'intermittent stiction': sarcomere groups that have released, are allowed to become fixed again at a later stage of the simulation (Fig. 5.8.).

In agreement with other authors (Matthews, 1972; Goodwin et al., 1975; Hunt, 1990), we assumed that stable cross-bridges re-establish spontaneously within intrafusal sarcomere groups, after they have first detached during muscle stretch. Also, we assumed that re-attachment of cross-bridges occurs at a spindle length different from the initial length. In this way, the linear range (of high length sensitivity) can establish itself at no matter what spindle length, depending on the muscle spindle's stretch history.

A time-constant for the re-establishment of intrafusal stiction is unknown (Hunt, 1990)[5]. We chose a time-constant which suited the model best (half of the released cross-bridges would re-attach after approximately 0.1s). It is beyond the scope of this chapter to describe in full detail the implementation of intermittent stiction within the muscle spindle model. Therefore, we only illustrate its effect by describing the modeled primary afferent response to a complex stretch regime of the following profile (Fig. 5.8.):

(1) a hold phase (at a muscle fibre length halfway its physiological range); (2) a ramp stretch (with an amplitude of 20% of the physiological range); (3) again a hold phase (at a muscle fibre length of 70% of its physiological range); (4) a ramp release of the same duration and of the same magnitude (but opposite sign) as the first stretch and, finally, (5) a hold phase at the starting spindle length. During each hold phase a very small test stretch (amplitude corresponding to 1% of physiological range) was imposed.

In figure 5.8a, the stretch regime, repeated twice, is illustrated by the heavy line. This figure also shows what proportion of the overall spindle length is taken up by the muscular part of the model's dynamic bag_1 fibre (continuous thin line) and to what extent this muscular part is 'fixed' by the presence of stable actin-myosin bonds (dotted thin line). Finally, figure 5.8b. illustrates the primary afferent response to the applied stretch regime.

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[5] Though recently such a figure is provided by Baumann and Hulliger (1991), suggesting that in the passive spindle cross-bridges are cycling, with a disruption and reattachment cycle of 1 s. This figure, however, became available after completion of the present study.

Starting off with a static discharge, the model's Ia-afferent activity responded with a steep rise in firing to the small test pulse applied 0.5s after onset of the stretch regime. This small stretch increased the intrafusal tension to such an extent that it surpassed the intrafusal breaking force F_x (0.045 arbitrary units: Schaafsma et al., 1991a). This caused fixed sarcomere groups within the dynamic bag_1 fibre to yield for about 50% before the intrafusal tension had decreased below F_x. With a time-constant ($t_{1/2}$) of approximately 0.1s the yielded sarcomere groups became fixed again, resulting in a rapid stiffening of the muscular part of the bag_1. When, at 1.1s after onset of the stretch regime, the ramp stretch was imposed, the spindle responded with a steep rise in firing activity (initial burst) followed by a dynamic response (which rapidly diminished once the active phase of stretching had ceased). During the ramp stretch, all sarcomere groups had yielded. They became fixed again straight after cessation of the ramp, but now at a larger spindle length than at the start of the simulation. Thereby, the linear range (of high spindle length sensitivity) became centred around the new spindle length.

When another test stretch was applied, the model again responded with a sharp burst of firing. The static discharge of the model, after the test stretch, was now lower than before. This was caused by the fact that the test stretch had caused intrafusal stiction to yield for about 75%. The length of the muscular part of the bag_1 fibre had briefly been allowed to increase and stiction re-established at this greater length. This caused a drop in static primary afferent discharge[6].

When the muscle spindle was released again, at 2.7s after onset of the simulation, the primary afferent firing dropped steeply due to unloading of the spindle's sensory regions[7]. It decreased further with decreasing spindle length. During unloading, the primary afferent firing was mainly based on the contribution of the static bag_2 fibre, since the muscular part of the bag_1 fibre was not able to keep up with the rapid decrease in spindle length and its sensory region

[6] For real muscle spindles such a small test stretch would not result in an observable change of static discharge. The static discharge may only be expected to drop after a large stretch or so-called 'killer stretch' which, in experiments, is used to abolish fusimotor after-effects. Apparently, the formulation of stiction within the muscle spindle model is too rigid in comparison with real spindles. As, in the model, no sway is allowed in the (virtual) myosin heads, the small test stretch immediately causes 75% of all cross-bridge regions to yield. Once yielded, these regions are allowed adapt to the present spindle length.

[7] In real muscle spindles, even when subjected to a small static fusimotor bias, it is expected that during a comparable release the primary afferent discharge would drop silent. This, in the spindle model, is still a question to be solved. Possibly, the maximal contraction velocity of the static bag_2 fibre in the model is set too large when compared with real muscle spindles. Or perhaps, peculiarities in the encoder properties may have to explain for the observed silencing during spindle unloading.

was fully unloaded. Under these circumstances we allowed part of the intrafusal sarcomere groups to detach again[8]. This allowed the slackened muscular part of the bag_1 fibre to 'contract' slowly.
While intrafusal tension in the bag_1 fibre was still being restored, two new test pulses were applied. Under these circumstances, the stretch could only be transmitted to the sensory region of the bag_2 fibre as the bag_1 sensory region was still fully unloaded. Consequently, the muscle spindle responded less vigorously. At 4.9s a renewed ramp stretch pulled the bag_1 muscular part taut again and allowed the length signal at the spindle poles to be transmitted to the its sensory region: on top of its dynamic response the model responded with a delayed initial burst. The remainder of figure 5.8. shows that from this point on, the model behaved quite similarly to the imposed stretch regime as described earlier.

The activity of the model's secondary afferents was derived from the spindle model's bag_2 fibre. Thereby, the static discharge of the secondary afferent was roughly equal to that of the primary afferent displayed in figure 5.7. However, the dynamic discharge of the secondary afferent was far less than that of the primary afferent.

A model of spinal reflex mechanisms.
Two reflex arcs (mediating proprioceptive input derived from three muscle spindle models, each within one of the three muscles governing the elbow joint) were implemented within the model of the elbow joint (Fig. 5.9.)[9]. One reflex arc was taken to provide postural rigidity, the other to provide postural plasticity.

Postural rigidity was achieved by implementation of the myotatic stretch reflex. This provided the model with a negative feedback system allowing it to correct for small perturbations of the arm. The existence of this reflex pathway is beyond doubt. We have not incorporated a reciprocal Ia-inhibitory reflex arc. On theoretical grounds it was argued that the effect of this

[8] By detaching part of the sarcomere groups, the intrafusal fibre is allowed take up slack and restore the intrafusal tension. In the model this results in a slow contraction (with a timeconstant of roughly 1.5s). It should be noted that this mechanism is completely different, with a different timeconstant, from the mechanism by which an intrafusal fibre is allowed to 'stiffen' when all sarcomere groups have yielded due to a preceding stretch.
[9] It would not be wise to implement more than two reflex arcs. Too many implemented reflex pathways would extremely complicate any interpretation of simulations provided by the model.

reflex pathway would be similar to that of the myotatic reflex, namely to increase postural rigidity: the reciprocal inhibition of the antagonist muscle by stretch of the agonist, would only cause a further decrease of antagonist muscle activity, on top of that expected on basis of its unloading reflex.

The mechanism by which postural plasticity occurs, is highly complicated (Angel, 1983; Schaafsma and Van Willigen, 1991c). It can theoretically be implemented by any reflex mechanism which provides inhibition to alpha motoneurones of a stretched muscle and disinhibition to the alpha motoneurones of a shortening muscle. Implementing such a pathway requires: 1. the choice of an appropriate receptor signalling a change of elbow joint angle and 2. the mechanisms underlying the eventual postsynaptic effect (facilitatory or inhibitory) of excited afferents onto spinal alpha motoneurones.

For practical reasons, we have decided to let the plasticity response be based upon an inhibitory pathway from muscle spindle secondary afferents to the homonymous alpha motoneurons, as suggested by Gregory et al. (1990), although data exist in literature which oppose the existence of such a reflex mechanism. Evidence has been gathered showing that muscle spindle II-afferents exert an excitatory action upon autogenetic alpha motoneurones, in addition to the excitation already derived from primary afferents (Matthews, 1969; Kirkwood and Sears, 1974; Stauffer, Watt, Taylor Reinking and Stuart, 1976).

Also, other possible candidates exist which may provide postural plasticity. For instance, the Ib inhibition pathway to autogenetic alpha motoneurones may prove to be such a candidate, especially now evidence has been forwarded that the Golgi tendon organs may also signal small changes in muscle tension (Binder, Kroin, Moore and Stuart, 1977)[10]. Nevertheless, whatever is the nature of the plasticity response, it was the purpose of the present study to demonstrate how this response may interact with the myotatic reflex without rendering the elbow joint unstable.

Alpha motoneurons were modeled as neurons receiving a background activation, added with the excitatory action of primary afferent activity and decreased by the inhibitory action derived from secondary afferent activity. The two feedback loops were delayed by latencies of 30 and

[10] Indeed, during this study several attempts were undertaken to derive postural plasticity from the well documented Ib-inhibitory loop. Apart from the uncertainty about the exact response characteristics of Golgi tendon organs (we did not dispose of a model predicting Ib-afferent discharge), these attempts were never very succesful. On stretch of a muscle, the increased muscle tension may cause an inhibition of alpha motorneurons, arriving almost at the same time as the Ia-afferent excitation. It is difficult to predict what will be the net result.

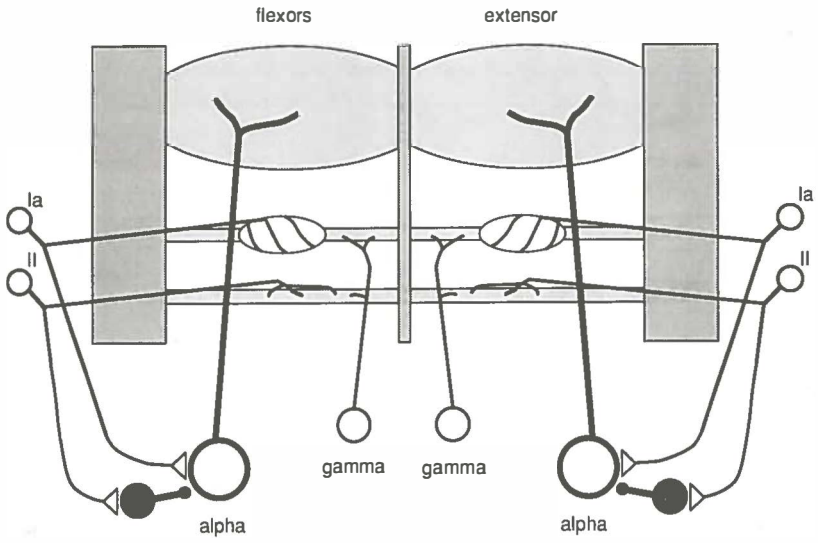

Figure 5.9.:

A schematic representation of the reflex arcs implemented within the arm model. The angle and stiffness of the elbow joint are controlled by two antagonistic groups of muscles (the elbow flexors, represented by the mm. biceps and brachialis, and its extensor, the m. triceps). A muscle spindle is situated in each of the three modeled muscles. With a small delay, primary afferent activity exerts an excitatory action on the autogenetic alpha motoneurons. With a relatively large delay,the secondary afferent projects to an inhibitory interneuron (not explicity modeled) which, in its turn projects to the autogenetic alpha motoneuron. The gain of the synaptic transmission in both pathways is chosen by trial and error. The fusimotor conditions, controlling the sensitivity of the simulated muscle spindles, remain unchanged.

300ms[11], respectively.

[11] The latter latency of 300 ms seems too large to be physiological. It is conceivable that the latency of the plasticity response should be larger than that of the myotatic reflex, in order to preserve joint stability. In agreement with the study of Schaafsma and VanWilligen, who showed that shortening and lengthening reactions occurred roughly 100 ms after onset of a perturbation, we started our simulations with a latency of 100 ms. The results obtained were not significantly different from those presented here. However, because of the little spacing in time, it was hard to demonstrate the effects of either reflex loops independently (e.g. in Fig. 5.14). For presentation reasons we chose a latency of 300 ms.

The strength of synaptic transmission was controlled by two gain factors (one of a positive sign for the primary afferent loop and one of a negative sign for the, inhibitory, secondary loop). These gain factors were the same for each of the three muscles modeled. Parameter values for the gain factors were chosen by trial and error until the model behaved to our satisfaction.

The dynamics of the forearm movement.

The dynamics of the model's forearm were simulated by iterative computations over short periods of time (δt). The value for δt was usually chosen 0.01 s for perturbation simulations and 0.001s for the calculations on joint stiffness. Moreover, it was always ascertained that the choice of a smaller timestep did not influence the results provided by the model.

For each step in time the contribution in torque was calculated for each muscle based upon (1) muscle activation; (2) muscle length at current joint angle; (3) muscle velocity derived from muscle lengths at current and previous joint angle and (4) lever arm at current joint angle. The torques exerted by the mm. biceps and brachialis were added to give a single negative torque while the torque exerted by the m. triceps was taken as positive. The torque exerted by forearm moment of inertia was taken as the product of this moment of inertia and the forearm acceleration (based on current and previous joint velocity). Once these torques were calculated, their net sum determined the forearm acceleration after a time-increment δt. Then, the forearm velocity and forearm position were calculated.

In fact, the method described above, by its dependence on the calculations of velocity and acceleration, was always two or three steps behind. Therefore, it tended to produce errors in calculation of the arm position, due to the finite δt. We were able to reduce such errors by use of the Newton-Raphson method (Press et al., 1986) with numerically determined first derivatives. At each timestep this method searched for that position of the forearm for which the net sum of muscle torques and inertial torque was zero. In this way estimates for velocity and acceleration were one timestep fewer behind.

After all free parameters of the model had been set, we adjusted the gain of the two feedback loops (the excitatory pathway from the Ia-afferents and the inhibitory pathway from the II-afferents, both projecting to the autogenetic alpha-motoneurons) by trial and error until the model behaved with a suitable mixture of rigidity and plasticity. Subsequently, the model was tested under similar conditions to those used in the experimental study on human subjects (Schaafsma and Van Willigen, 1991b/c).

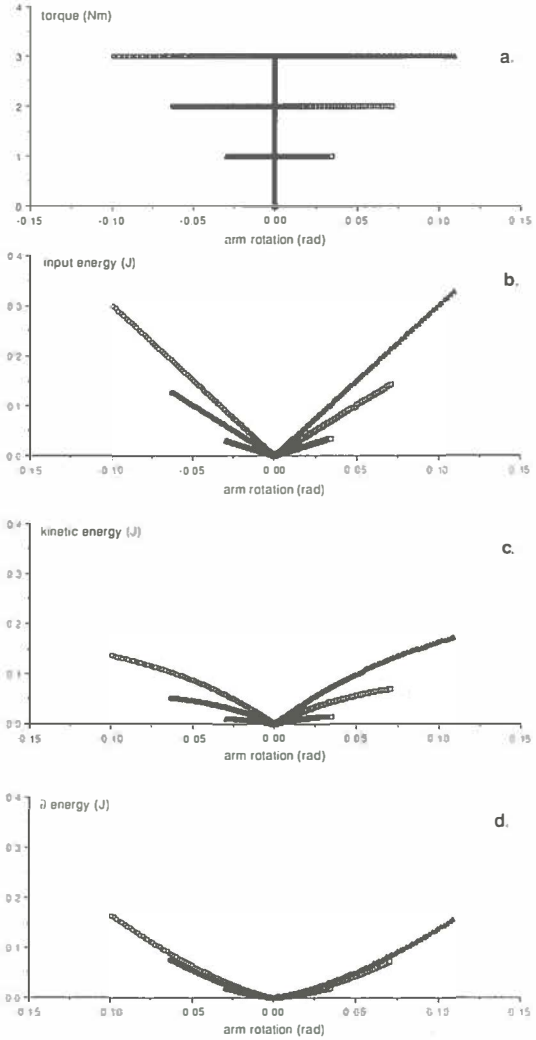

Figure 5.10.:

a. Model simulation of the arm when 6 perturbations are imposed (torques of -3, -2, -1, 1, 2 and 3 Nm, duration 0.1 s). The torque is plotted as function of the resulting arm rotation.
b. Energy put into the model's arm, obtained by calculating the surface below the plots in fig. a.
c. Kinetic energy of the arm as function of arm rotation.
d. Amount of energy absorbed by the visco-elasticity of the model's muscles as a function of arm rotation (δ energy). This was calculated by subtracting the kinetic energy (c.) from the total input energy (b.).

In the first place, our aim was to determine the mechanical joint impedance of the model by imposing perturbations to the model's forearm and making a similar analysis in the energy domain as in the experimental study. It was of interest to investigate how the calculated joint impedance depended on the level of antagonistic co-contraction. Secondly, we were interested to see how the model achieved posture control, now it had been tuned to respond to passive movements with a mixture of postural rigidity and plasticity.

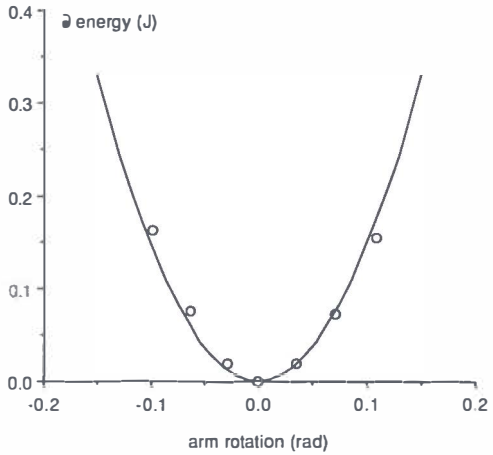

Figure 5.11.:

Model outcome of the final values for δ energy calculated 100ms after start of a simulated perturbation. These data are independent of postural reflexes due to the latency of spinal reflexes and electro-mechanical delay (the delay between muscle activation and the development of muscle force; Otten 1987b). A second order parabola is fitted to the data and also drawn in this figure. The calculated stiffness amounts 29.4 Nm/rad.

The calculation of joint stiffness and its relation to the level of antagonistic co-contraction.

The mechanical joint impedance of the human elbow joint was determined by measuring the torque required to move the forearm as a function of arm displacement. This mechanical joint impedance was highly dependent on the perturbation velocity, partly because of forearm moment of inertia and partly because of muscle viscosity[12]. In order to determine to what extent

[12] It should be stressed that we have used the term muscle viscosity to designate the velocity dependent muscle force. The muscle viscosity is not a constant but is a function of its contraction velocity.

the arm muscles contributed to the overall joint impedance it was necessary to subtract torques occurring because of the forearm's moment of inertia. These torques were calculated by multiplication of this moment of inertia with the forearm acceleration.

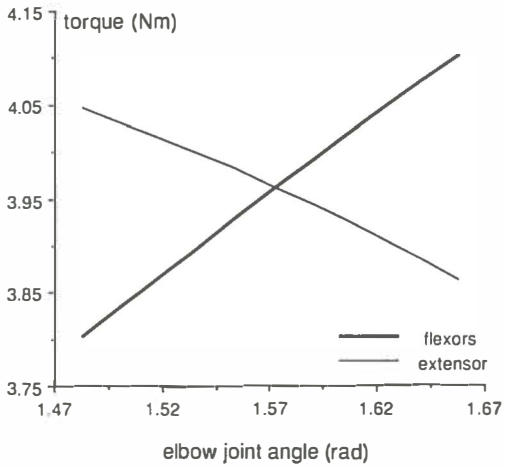

Figure 5.12.:

High resolution plot of the torque-angle relationship for the model's elbow flexors and extensor at the muscle activations maintaining the unperturbed arm at 90 degrees flexion at the elbow. Out of this plot a joint stiffness can be calculated which is only based on muscle elasticity. The elastic contribution to overall joint stiffness amounts 2.7 Nm/rad which is only 10% of the total joint stiffness calculated in figure 5.11.

In the experimental study, however, the determination of forearm acceleration, being the second derivative of arm position, was blurred by the inevitable amplification of noise in the position signal. Therefore, we chose to analyse joint impedance within the energy domain; instead of making use of a calculated forearm acceleration we used the forearm velocity, which was less blurred by noise as it was based on the first derivative of arm position.

5.3. RESULTS

We were interested to see whether the calculation of joint visco-elasticity out of joint impedance, as extracted from our experimental data, proved correct for the arm model also. We imposed similar perturbations to the model's arm as in the experimental study (maximal motor torques in

the experimental study amounted 2.5 - 3.0 Nm) and processed the data on arm rotation and velocity in the same way as the experimental data (Fig. 5.10.). Figure 5.10a. illustrates the perturbation torques imposed on the model's arm as a function of arm rotation (arm rotation as function of time is illustrated by Fig. 5.16a.). These torques were kept constant throughout the perturbation and were of the same order of magnitude as in the experiments. As

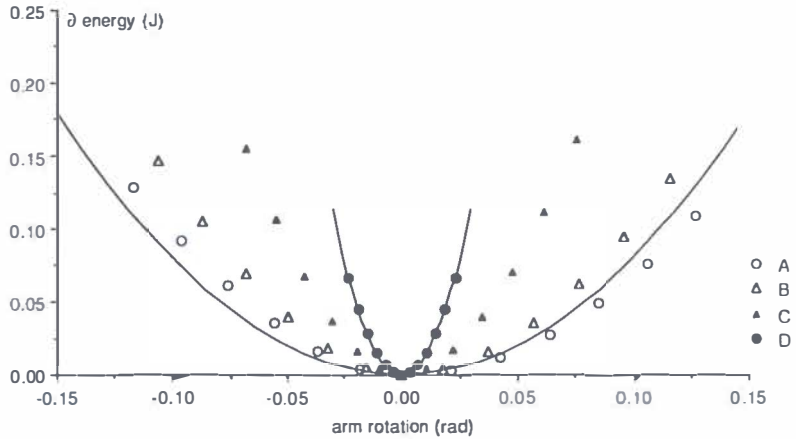

Figure 5.13.:

Figure illustrating the dependence of the model's joint stiffness (as calculated in the energy domain) for different levels of antagonistic co-activation. Flexor muscles were activated at 0, 5, 10 and 20 % of maximum for A, B, C and D; activation levels for the m. triceps were adjusted in order to maintain the model's elbow joint at an angle of 90 degrees flexion and amounted to (in the same order): 7.6, 9.0, 13.5 and 27.0 % of maximal activation. We fitted second order polynomials through the data obtained for A and D. By doing so, we derived a joint stiffness of 16.0 Nm/rad for A and of 251.4 Nm/rad for D.

in the experiments, we only took the first 100 ms of each perturbation, a time interval during which muscle activations may already have changed but for which no change in muscle torque was yet expected due to the muscles' electro-mechanical delay.

The amount of input energy (Fig. 5.10b.), delivered by the perturbation apparatus to the arm, was obtained by integrating the surface below the plots of torque versus arm rotation. As the perturbation torques were constant, the input energy as function of arm rotation followed a straight line through the origin, the steepness of which depended on the magnitude of the

torque. Part of the input energy was used for the acceleration of the forearm, it was transformed to kinetic energy (Fig. 5.10c.) of the moving inertial element. This kinetic energy could be calculated from the forearm's moment of inertia (0.09 kg·m) and from the arm velocity.

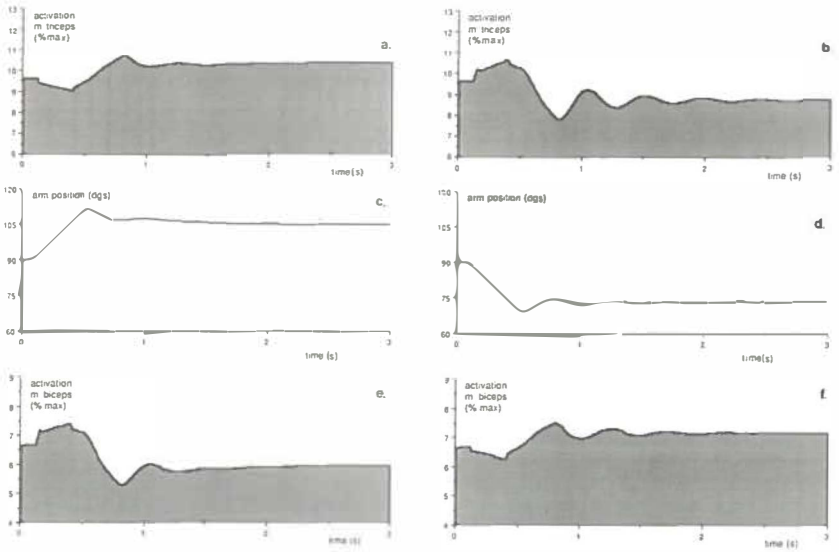

Figure 5.14.:

Outcome, simulated by the model, of the activations of m. triceps (a and b) and m. biceps (e and f) in response to an imposed 20 degrees extension (c) or flexion (d) of the model's arm. In both cases the stretched muscle responds with an initial increase (myotatic stretch reflex), followed by a sharp and tonic decrease in activation (lengthening reaction). The released muscle, on the other hand, initially responds with a decrease (unloading response) which is followed by an increase in activation (shortening reaction). The combined effect of the shortening and the lengthening reaction cause the arm to settle at a new position, shifted in the direction of the imposed perturbation.

The remaining part of the input energy (δ energy) was absorbed by muscle elasticity and viscosity (Fig. 5.10d.).The δ energy was derived by subtracting the kinetic energy from the total input energy.

In order to determine one value for the model's joint visco-elasticity we plotted the data on δ energy attained 100 ms after onset of each perturbation (Fig. 5.11.). As in the experimental

study, we fitted these data with a second order polynomial. Such a dependence of δ energy upon joint angle would be expected if both antagonistic muscle groups behaved as simple linear springs. In this way we calculated a joint visco-elasticity for the model of 29.4 Nm/rad. This value falls close to the range of experimentally obtained visco-elasticities (Schaafsma and Van Willigen, 1991b).

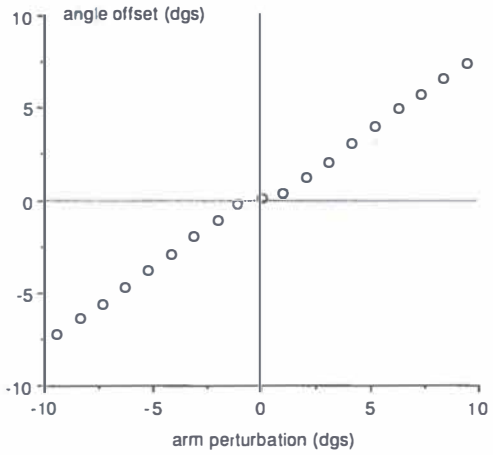

Figure 5.15.:

Plot illustrating the shift in the model's maintained arm position after different amplitudes of perturbation. The velocity of each perturbation varied, as the perturbation was constantly kept at 0.4s. The median plasticity ratio (defined as the difference between final and starting position of the arm divided by the maximally attained perturbation amplitude of the arm) is roughly 75%. There is a small interval around 0 degrees perturbation amplitude, for which the arm returns more accurately to the starting position. This interval depends on the width of the muscle spindle model's linear range and, thus, on the model's crossbridge breaking force.

It was interesting to see that in the model most of this calculated joint visco-elasticity was dependent on muscle viscosity and not on muscle elasticity. This becomes clear when a high resolution plot is made of the torque-angle relationship of figure 5.6. for the muscle activations maintained by the model in order to keep the arm 90 degrees flexed (Fig. 5.12.). From this figure the elastic (velocity independent) contribution of the modeled arm muscles could be deduced: for a small extension of the arm the flexor torque would increase by about 1.7 Nm/rad

and the extensor torque would decrease by approximately 1.0 Nm/rad. Consequently, we would expect a joint elasticity (or stiffness) of 2.7 Nm/rad, whereas the joint visco-elasticity was ten times as large. Apparently, muscle viscosity was more important for the joint visco-elasticity than muscle elasticity (this could also be ascertained by inactivating the model's force-velocity relationship and recalculating the muscle dependent joint stiffness).

Finally, we determined how the joint visco-elasticity varied for different levels of antagonistic muscle activation. For this purpose, we turned the gain of the two feedback loops to zero. We chose a muscle activation for the two flexor muscles (m. biceps and brachialis) and, subsequently, adjusted the level of triceps activation in order to let the model maintain the elbow at 90 degrees flexion. We calculated the δ energy for each combination of flexor and extensor activation and for the same perturbation torques as used in figure 5.10. Figure 5.13. shows how the joint visco-elasticity increases with increasing co-contraction. Again, we calculated the best second order polynomial fit through the data points of two of the tested combinations of antagonistic co-contraction. The calculated visco-elasticities ranged from 16.0 (flexors 0%, extensor 7.6% of maximal activation) to 251.4 Nm/rad (flexors 20% and extensor 27% of maximal activation). Again, joint visco-elasticity was mainly due to muscle viscosity and not to muscle elasticity. It was even found that at the highest level of co-contraction tested (combination D in Fig. 5.13.) the equilibrium point at 90 degrees elbow angle was unstable, accompanied by a negative contribution of joint stiffness to the overall joint visco-elasticity.

Postural rigidity and plasticity.

Figure 5.14. shows the response of the model to two perturbations: one of 20 degrees extension (Figs. 5.14a/c/e.), and one of 20 degrees flexion (Figs. 5.14b/d/f.). When considering the two position traces provided by the model (Figs. 5.14c/d.), it became clear that the model simulated the same plastic behaviour as observed in most of the subjects studied experimentally (Schaafsma and Van Willigen, 1991c). It should be noted that the arm angle did not change any further beyond the interval of three seconds that has been displayed.

During a passive extension, the modeled m. biceps (Fig. 5.14e.) initially responded with a sharp rise in activation due to the initial burst and dynamic response in primary afferent firing derived from its stretched spindle model. After a delay of 0.3 s this increase in biceps activation was suppressed by the inhibition derived from a gain in firing of the secondary afferents. In doing so, the model simulated the so-called lengthening reaction observed in EMG-recordings

of our subjects. By its decreased activation, the m. biceps gave in to the imposed perturbation and allowed the arm to become stabilized at its new joint angle.
At the other side of the joint, the m. triceps responded with opposite changes in activation:

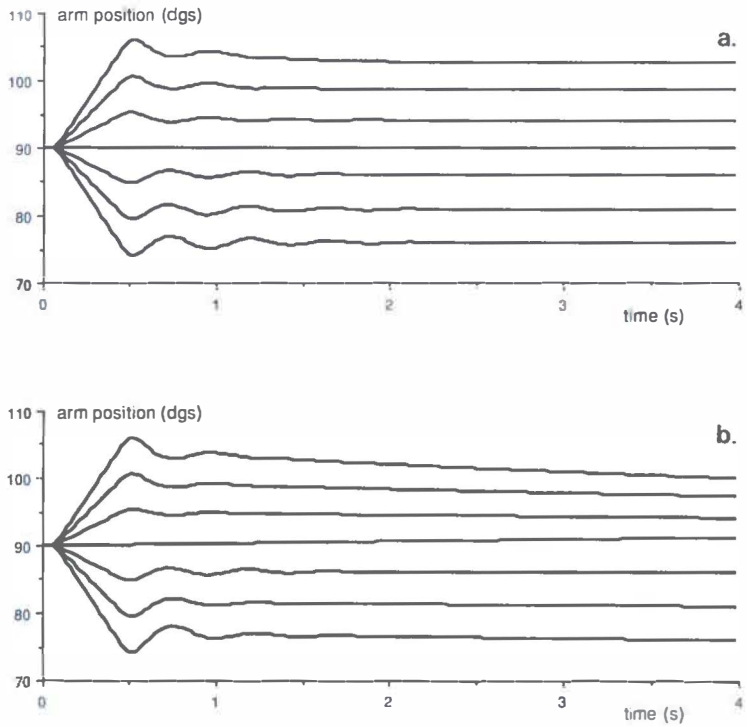

Figure 5.16:

The effect of stiction on the stability of arm position when 6 perturbations and one control (torques of -3, -2, -1, 0, 1, 2 and 3 Nm; duration of 0.4 s) are imposed on the model's arm, either with stiction in the modeled muscle spindles (a.; crossbridge breaking force $F_x = 0.045$) or without stiction (b.; $F_x = 0$). With stiction, the model's arm rapidly comes to a rest after a perturbation and maintains its newly acquired position for the full remaining of the illustrated period of time; whereas, without stiction, the arm starts to drift. Note that postural drift even even occurs when no perturbation is imposed (middle trace).

during the first 0.3s of the perturbation, the muscle activation decreased as result of spindle unloading, whereas, after this period of time, the muscle rapidly gained in activation due to a decrease in secondary afferent inhibition. In EMG-recordings this behaviour is known as the so-called shortening reaction. By increasing its activation, the m. triceps gained in force and helped to maintain the arm at its new position.

While, in response to an imposed extension, the lengthening reaction in the m. biceps and the shortening reaction in the m. triceps caused the arm to shift its maintained posture towards extension, the opposite occurred in response to an imposed flexion of the arm: a shortening reaction of the m. biceps (Fig. 5.14f.) and a lengthening reaction of the m. triceps (Fig. 5.14b.) caused the arm to shift its posture towards flexion (Fig. 5.14d.).

When we imposed a series of perturbations on the arm model and plotted the shift in maintained elbow joint posture as function of maximal perturbation amplitude, we obtained a graph similar to those obtained in our experiments (Fig. 5.15.). The plasticity ratio for the model, defined as the ratio of final arm position and maximal perturbation amplitude (Schaafsma and Van Willigen, 1991c), is roughly 75%. Only for the interval of -1 to +1 degrees perturbation amplitude does the arm return close to its original position. It could be shown by varying F_x that the width of this interval is dependent on the choice for the intrafusal breaking force. The larger F_x, the wider the interval for which the arm returned to its original position (not illustrated)[13]. A side-effect of increasing the intrafusal breaking force F_x, however, was that the model's arm tended to start oscillating around its resting position, especially for the more flexed arm postures. An increased F_x, apparently, induced postural oscillation by enhancing the range of movement amplitudes for which stretch reflex gain was maintained high, owing to persisting high peripheral spindle gain.

Another illustration of how intrafusal stiction in the model's muscle spindles may add to the stability of arm posture is illustrated in figure 5.16. By implementation of intermittent stiction, fixed intrafusal sarcomere groups within the muscle stretched during a perturbation were able to yield at first but re-establish later. The breakage and re-attachment of intrafusal crossbridges allowed stiction to yield at one spindle length and re-establish at a second and larger length. In the slackening muscle, on the contrary, the muscular part of the bag_1 intrafusal fibre was

[13] That a larger breaking force was required in the model of the human elbow joint as compared with the muscle spindle model, may be attributed to differences in the general build of human triceps and biceps muscles versus cat soleus muscle. Alternatively, another possible explanation is that in reality, a smaller fraction of the total change in muscle length is transmitted to the spindle poles than is assumed in the present simulation.

unloaded and, therefore, allowed to contract. In this way, stiction, from being centred around the starting length, shifted to a new and smaller spindle length. The combined action of these two mechanisms was that the newly acquired arm position was rapidly fixed by the rebuild of stiction within the muscle spindles of both antagonistic muscle groups. For the model, intermittent stiction was important in stabilizing the arm and preventing it from drifting away. This can be seen by comparing the behaviour of the model with (Fig. 5.16a.) and without stiction (Fig. 5.16b.).
Figure 5.16a. shows that with the occurrence of stiction within the model's muscle spindles, the model was well capable of stabilizing its posture at various angles of the elbow joint. In contrast, figure 5.16b. shows that without stiction (by changing to zero the intrafusal breaking force F_x) the model does not actually achieve stabilized joint angles. Instead, even without any perturbation imposed to the arm, the model displays a slow drift in elbow posture caused by minimal differences in antagonistic muscle torques.

5.4. DISCUSSION

In experimental studies at our laboratory it is shown (Schaafsma and Van Willigen, 1991b/c) that posture maintenance at the human elbow joint can be characterized in terms of rigidity as well as plasticity: in response to a perturbation and depending on the perturbation amplitude, a subject's arm may respond rigidly and return to its original posture as well as respond plastically and assume a new posture. This observation has led us to hypothesize that two different pathways exist for the processing of proprioceptive information by the central nervous system: the first pathway is the myotatic stretch reflex which provides the postural system with rigidity; the second pathway allows the system to respond plastically. By means of a computer model, we have aimed to investigate whether the simultaneous action of these two pathways would indeed lead to the experimentally observed mixture in postural rigidity and plasticity.
In order to be able to draw conclusions out of the model's results it is necessary to bring under discussion the reliability of the model. A large part of this section is, therefore, dedicated to considerations about the validity of approximations and assumptions which were necessary for the formulation of the model. After establishing the reliability and limitations of the model, some conclusions are made on basis of the model's results. However, before all of this, some general remarks need to be made upon the philosophy which underlies our modelling attempts.

General remarks.

Many experimentalists overestimate the implications which a modeler wishes to make on basis of his/her model. This often causes them to enquire whether the model can sufficiently describe and explain highly detailed experimental observations. If the model does not satisfactorily predict these observations, they conclude that the model cannot be trusted and, therefore, cannot teach us anything about reality. In our opinion, such an approach does no right to the great advances in understanding that can be achieved by the modelling of physiological processes.

We are convinced that the only way to evaluate the action of two or more physiological mechanisms acting simultaneously on one biological parameter is by means of (computer) simulation. The unaided human mind is very poor in such an evaluation: in the laboratory we attempt to isolate physiological mechanisms under study as much as possible. But even when a full definition of input-output relations is reached for one mechanism, all these conclusions become uncertain when considering a second. In such cases a model simulation may help to understand how two or more hypothetical mechanisms may act in concert and what their relative contribution is to the overall behaviour.

Nevertheless, a model can never meet the high dimensionality of real physiological systems, since it generally makes use of simplifications, assumptions and omissions. Therefore, we feel that a model should not be seen as the full description of reality. From this point of view, a model can be no more than a quantitative illustration of how the modeler envisages reality.

Considering the model presented in this chapter, it should be considered as a quantitative description of how two mechanisms, with opposite effect in response to muscle stretch (postural rigidity and postural plasticity), may co-exist and co-operate. In addition, the model shows how specific properties of muscle spindle sensitivity may add to the stability of the postural system and prevent the occurrence of slow postural drift. Keeping in mind these limited objectives, we will now discuss the assumptions, simplifications and omissions made during the formulation of the model. Similar paragraph titles will be used as in the methods section.

A model of forearm and elbow joint.

The human elbow joint was modeled as a cylindrical joint (Fig. 5.1.). This is correct only in approximation. It is known that changes in elbow angle will be accompanied by a conjoint rotation of the forearm. We have ignored effects of any forearm pronation or supination (for

instance on the fibre lengths of the modeled muscles). Such a simplification seemed reasonable for the limited range of elbow angles studied (within a range of 50 to 130 degrees joint angle). Also, in the experimental studies the fixation of the arm did not allow any rotation of the forearm.

Accepting the simplification that the elbow joint is cylindrical, any movement around the joint can be described in terms of a simple rotation. When the rotational axis of the joint is kept perfectly perpendicular to the horizontal plane any movement of the forearm will take place without work done by gravity.

A model of the muscles governing the elbow joint.

The angle and stiffness of the modeled elbow joint was controlled by two antagonistic muscle groups. We have only considered the m. biceps and brachialis as elbow flexors and the m. triceps as elbow extensor. These muscles describe the system adequately, since the other elbow muscles (mm. brachio-radialis and anconeus) have comparable muscle vectors and are much weaker as estimated from their physiological cross-sectional area.

The muscles were all represented by single force vectors, compartmentalization of the muscles was not taken into account. Also, we ignored that the m. biceps and triceps are, at least partially, bi-articulate. This simplification is justified as, in the experimental studies, the angle of the shoulder joint was kept constant. Without a change of shoulder angle, the position of the muscles' origins was constant with respect to the humerus. Changes in muscle fibre length, therefore, only occurred on basis of changes in the angle of the elbow joint.

Finally, the direction of the force vectors was always assumed to be in parallel with the humerus. This is only true in approximation. The changing lever arms of the muscles will cause their force vectors to shift in direction. Effects of these shifts are not great, as the distance between the muscles' origins and insertions is relatively large compared to the change in lever arms.

Though changes in muscle lever arm with changes in joint angle were judged less important for the direction of the force vectors, they largely influenced the magnitude of the muscle torques. An et al. (1981) measured muscle lever arms at three different angles of the elbow joint. Therefore, we had to extra- and interpolate these data for other elbow angles (Fig. 5.2a.). This was achieved by means of a second order polynomial fit. For the m. triceps and brachialis the polynomial fit resulted in muscle lever arms which increased and decreased as monotonic

functions of joint angle; for the m. biceps a maximal lever arm was attained at approximately 70 degrees elbow angle.

Muscle lever arms for the mm. brachialis and biceps are also modeled by Van Zuylen et al. (1988). Their data agree with ours on a maximal lever arm for the m. biceps at 70 degrees elbow angle. However, Van Zuylen et al. also found a maximal lever arm for the m. brachialis at an elbow angle of roughly 90 degrees, whereas our simulation only provided a maximal lever arm at full arm flexion. The absolute values for the muscle lever arms calculated by Van Zuylen et al. (1988) differ by a factor of two from those measured by An et al. (1981). Possibly, these differences are caused by differences in the subjects' body size. The study of Van Zuylen et al. does agree with An et al. that the lever arm of the m. biceps is one and a half times larger than that of the m. brachialis. We were unable to use the model of Van Zuylen et al. for the present simulation as it did not contain any information about the m. triceps.

From the muscle lever arms we calculated the change in muscle fibre length as function of joint angle (Fig. 5.2b.). The method used was a crude one but reasonably accurate for joint angles close to 90 degrees. It resulted in muscle fibre lengths which increased (mm. biceps and brachialis) or decreased (m. triceps) as monotonic functions of joint angle[14].

We fed the calculated muscle fibre lengths into the dynamic muscle model developed by Otten (1987b). However, with respect to the arm model there were still a number of free parameters, the values of which we extracted from literature where possible. Yet, a number of parameters remained arbitrary. Their effect on the simulated muscle force can best be evaluated by considering the force-length relationship provided by figure 5.4a. The arbitrary parameters concerned the localization of the maximal active muscle force (optimal length) and the steepness and localization of the passive muscle force (c_1 and c_2). The values of the latter parameters differed from those suggested by Otten (1987b) because the arm model disposed of muscle fibre lengths, whereas in the paper of Otten the values c_1 and c_2 were adapted to full muscle length, including muscle tendons. A different choice of parameter values may certainly cause changes in the torque output as function of joint angle. Nevertheless, it is not expected that such changes would alter the (limited) conclusions of this study.

[14] This is only true when it is assumed that the muscle tendons behave with zero compliance. It should be noted that a large proportion of the total muscle length, in particular of the m. biceps and triceps, is taken up by the tendon, of which at least some compliance should be expected.

A model of receptor activity.

The model for muscle spindle primary afferent firing which was described previously (Schaafsma et al., 1991a), was altered in two places: firstly, a different choice was made for the breaking force F_x and, secondly, intrafusal stiction was made dynamic (when yielded, stiction could re-establish at any given spindle length). The latter change proved to be a key expansion of the model to improve postural stability.

We chose a value for F_x of 0.045 (in arbitrary units) since it gave workable values for the muscle spindle's intermittent stiction: it caused the intrafusal stiction to yield (by forcing stable actin-myosin bonds to break) as soon as the forearm was rotated more than 0.8 degree.

Intrafusal stiction is held responsible for the linear range in muscle spindle sensitivity to sinusoidal stretching as well as for the initial burst in the spindle response to ramp-and-hold stretches (Matthews, 1972; Hasan and Houk, 1975; Proske and Stuart, 1985). Due to intrafusal stiction, the sensitivity for a small ramp change in length may become as large as 280 pps/mm (Hasan and Houk, 1975). Several studies indicate that intrafusal stiction may occur at any spindle length and is highly dependent upon the past history of spindle length (e.g. Matthews, 1972; Goodwin et al. 1975; Gregory et al., 1990).

How intermittent stiction is implemented within the muscle spindle model is not described in detail in this chapter. Instead, we provided a graphical impression of the spindle model's behaviour in response to a complicated stretch regime (Figs. 5.8a/b.). Since we lacked detailed experimental information on intermittent stiction, the model's time-constants (underlying the rebuild of cross-bridges and the speed with which a slackened intrafusal fibre catches up with a shortening muscle spindle) were a source of concern[15]. Hunt (1990) reports that after a large stretch, full recovery of the muscle spindle's linear range may not occur before nearly 15 s, whereas Matthews (1981) estimates that intrafusal stiction can recover within 1 second. In the model, full recovery of stiction, at least for the slackening muscle, roughly takes 2.5 s. The shorter this period, the more rapid the arm came to a stand after a perturbation.

All considerations about intrafusal stiction concerned the model's primary afferent only. The plasticity response made use of a II afferent activity derived from the spindle model's bag_2 fibre. This activity was not further tuned to experimental data as it was multiplied with an arbitrary feedback gain anyway. The II-afferent feedback did not display any effects of intrafusal stiction.

[15] New information on this subject (Baumann and Hulliger, 1991) became available too late for incorporation in the model presented.

A last simplification was that fusimotor conditions were assumed constant; we chose a static gamma bias of 20 Hz and a dynamic gamma bias 0 Hz. Turning the dynamic fusimotor conditions to zero (in the spindle model) improved the effect of intrafusal stiction (Schaafsma et al., 1991a). This is, most likely, not the case for real muscle spindles. On the contrary, the high spindle sensitivity to small changes in length may become increased as an after-effect of dynamic fusimotor stimulation (Brown et al., 1969; Hulliger et al., 1977). The choice for 20 Hz static fusimotor activity was arbitrary and caused the model to be slightly more length sensitive than it would have been under passive circumstances.

A model of spinal reflex mechanisms.

Two reflex mechanisms were part of the model of posture maintenance at the human elbow joint: (1) the myotatic reflex and (2) a hypothetical II-afferent inhibitory pathway to the autogenetic alpha-motoneurons, based on a suggestion of Gregory et al. (1990). These authors investigate H-reflexes and myotatic stretch reflexes in the ankle flexors of human subjects. They find that the myotatic reflex of the soleus muscle decreases when, prior to testing, this muscle is contracted actively at a length larger than that of testing; whereas the amplitude of the H-reflex is increased by this same procedure. Gregory et al. attribute the observed changes in activity of the myotatic stretch reflex to a yield of intrafusal stiction after the conditioning contraction. Also, they suggest that the increase in H-reflex activity on muscle stretch is brought about by shifts in secondary afferent activity associated with pre-synaptic inhibition. For this chapter, we consider the effect of a direct inhibition of alpha motoneurons by autogenetic secondary afferents (Harrison and Jankowska, 1985).

II-afferent inhibition of autogenetic alpha motoneurons is controversial. Matthews (1969) in the decerebrate cat, suggests that, in response to muscle stretch, secondary afferents cause excitation of homonymous alpha motoneurons. This has been verified in later studies by several other investigators (Kirkwood and Sears, 1974; Stauffer et al., 1976).

Houk (1979) suggests an inhibitory role for the Ib-afferents. He reaches a conclusion similar to ours: neither muscle length nor muscle force is the parameter uniquely controlled by the central nervous system. Houk suggests that a combination of these two, namely muscle stiffness, is the actual parameter controlled by the nervous system. We made several attempts to bring into our model the conjoint action of Ia- and Ib-afferent feedback loops, but reached the conclusion that their simultaneous action does not easily lead to a stable arm posture: at a constant muscle

length, the inhibitory feedback from Ib-afferents to autogenetic alpha motoneurons easily introduces oscillation.

Another well known spinal reflex mechanisms which have not been considered in the present study are the Ia reciprocal inhibition and the Renshaw inhibition. Of these two, the first is likely to increase postural rigidity: during stretch of the agonist muscle, the reciprocal inhibition would only further decrease the activity of the antagonist muscle, on top of the decrease expected on basis of an unloading response. What is the effect of Renshaw inhibition in the presented arm model is difficult to predict. Future simulation experiments may help in disclosing what is the functional significance of recurrent inhibition.

In our arm model, the action of the Ia afferent loop was delayed with 30 ms and that of the II-afferent feedback loop with 300 ms. The first figure is in agreement with spinal reflex latencies. The long latency of the latter response is arbitrary due to uncertainty about the nature of the plasticity response (Angel, 1983). The value of this latency, however, did not appear critical: for 100 ms the arm stabilized equally well at positions shifted with the direction of imposed perturbations. The arm only stabilized more abruptly and displayed less damped oscillations as familiar from the experimental study. For presentation reasons, we preferred a latency of 300ms, so that the effect of either reflex loop could easily be demonstrated in the relevant figures.

Conclusions

The question to what extent myotatic reflexes add to the maintenance of posture is difficult to answer. According to Sherrington (1909), myotatic reflexes are apt to restore posture once it is perturbed.

Marsden et al. (1981) and Nashner (1976, 1983), however, show that, after a perturbation of physiological magnitude, the postural system responds with a set of reflexes, of variable amplitude and latency. Rack (1970) has emphasized that, because of these reflex latencies, antagonistic muscles controlling joint posture stand alone during the first moments of a perturbation. He suggests that intrinsic muscle properties, such as muscle stiffness, are of far greater importance for the maintenance of posture than the delayed action of short, medium or long latency stretch reflexes. Also, Otten (1987b) has emphasized the importance of force-velocity characteristics of muscles for the immediate resistance against a perturbation. Finally, this point of view is supported by reports that the gain of the myotatic reflex is not strong enough to antagonize successfully a major change in load (Evarts, 1985; Matthews, 1966).

Apparently, the output of the stretch reflex is not quick and not great enough to compensate for large changes in maintained joint posture. This is in line with our own findings: large perturbations are followed by a shift in maintained arm posture (Schaafsma and Van Willigen, 1991c) and small and abrupt perturbations are mainly antagonized by the forearm's moment of inertia and the intrinsic properties of the elbow muscles and not by reflex changes in muscle activation.

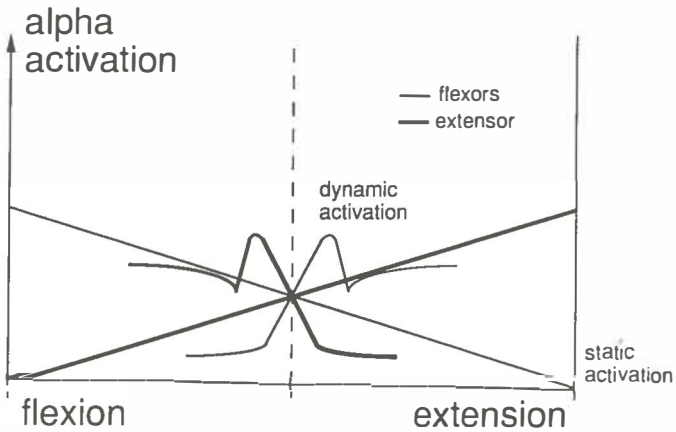

Figure 5.17.:

The combined effect of postural rigidity and postural plasticity, as simulated in the arm model. The gain of the model's inhibitory feedback loop from secondary afferent to autogenetic alpha motoneuron is adjusted in order to overrule the static excitatory action of the feedback loop from primary afferent to the autogenetic alpha motoneuron. This is the hypothetical basis of the plasticity response. Nevertheless it causes the arm to become unstable (see text). The postural system is prevented from this instability by stiction effects in the model's muscle spindles. The absolute and high length sensitivity of the model's muscle spindles, brought about by stiction effects within the dynamic-bag$_1$ fibre, causes the alpha activation to increase in response to a small shift in posture. In this way, intrafusal stiction possibly serves a delicate posture control: small changes in elbow posture are corrected for, larger movements will cause the stiction effects to disappear and allow the arm to assume a new and different posture.

A co-contraction of elbow antagonists increases joint visco-elasticity (Schaafsma and Van Willigen, 1991b). In the present study it is shown that this joint visco-elasticity is mainly determined by velocity dependent muscle properties (force-velocity relationship, Fig. 5.4b.) and not by the muscles' elastic properties (force-length relationship, Fig. 5.6.). In the model,

the muscular contribution to the total joint visco-elasticity amounted 29.4 Nm/rad. Of this 29.4 Nm/rad, only 2.7 Nm/rad was derived from joint elasticity, per definition independent of muscle velocity. So, 90 % of the apparent joint stiffness depends on velocity dependent muscle force. This suggests a re-evaluation of the explanation of Humphrey and Reed (1983) for the rise in joint impedance found after an increase in antagonistic co-contraction: the rise in joint impedance on increased antagonistic co-contraction is mainly caused by the gain in muscle viscosity (Hill, 1938; Aubert, 1956) and not due to a rise in the steepness of the muscles' length-tension relationship.

Though myotatic reflexes may be of little use for antagonizing suddenly imposed perturbations, they may be crucial for the prevention of slow drift. To us, this seems the main role of myotatic reflexes in posture maintenance at the human elbow joint. Only a small gain is required for such a control system, which adds to the prevention of postural oscillation. Moreover, the myotatic reflex is capable of responding to minute changes in muscle length, due to its high length sensitivity. As this high sensitivity is based on the yield and re-establishment of intrafusal stiction, it can disappear during movement and re-occur at any static joint posture.

The effect of intrafusal stiction can also be explained by means of figure 5.17. Let us assume that the arm is held at the mid position of the y-axis of this figure. Now imagine a small drift of the arm towards extension. This small drift will (on basis of the plasticity response) be accompanied by a definite decrease in flexor activation (straight, heavy line) and an increase in extensor activation (thin line). If these changes in muscle activation are strong enough (and they should be, in order to make the arm plastic) they result in a drop in flexor torque, as well as in a rise of extensor torque. The minimal extension of the arm will, therefore, be enhanced by the induced changes in muscle activation: the arm will drift further and further until it reaches maximal extension. A similar chain of events will cause the arm to move towards maximal flexion, if by chance, the first minimal drift was in that direction.

Now, this slow drift can be prevented by introduction of intrafusal stiction. The high length sensitivity of the model's muscle spindles will centre itself around the current joint angle. Instead of a decrease, the same minimal extension of the arm will now cause an increase in flexor activation. So shall a minimal flexion of the arm cause an increase in extensor activation. Thus, for small deviations of the arm posture from its setpoint, the myotatic stretch reflex will direct the arm back to its original position. For large deviations of the arm posture from its setpoint, the high length sensitivity due to intrafusal stiction will disappear and only re-occur when the arm comes to a rest at a new position.

That intrafusal stiction in the model leads to a stabilization of joint posture is illustrated by Fig. 5.16. In this figure the behaviour of the arm model is displayed during and after a set of perturbations. In figure 5.16a. the model's muscle spindles make use of intrafusal stiction. The arm is effectively stabilized at any given joint angle. In figure 5.16b., however, where stiction is removed from the arm model, the joint angle starts drifting, even when no perturbation is imposed. In comparing these two figures it becomes clear that intermittent stiction may help to stabilize any given joint posture.

The computer model had difficulties rendering an arm posture stable as it calculated muscle torques with a 'precision' up to twenty decimals: any inaccuracy in the calculated equilibrium results in a drifting joint angle. Without the introduction of the intrafusal stiction phenomenon, it is hard to imagine how the computer would be able to find the correct muscle activations resulting in equal opposing flexor and extensor torques.

The central nervous system must have comparable problems in balancing flexor and extensor torques, it has to deal with many sources of non-linearity: changing muscle lever arms, changing directions of the muscles' force vectors, an elbow joint which is not perfectly cylindrical, small changes in gravitational force, non-linearities in the muscle force-length relationship, etc. Intermittent stiction occurring in muscle spindles of a postural system may render stable any given joint angle despite such non-linearities.

CHAPTER 6

Conclusion

At the end of this PhD-thesis, it is useful to summarize the main conclusions of this study:

1. there is more to posture maintenance than the reflexes occurring in response to a perturbation,
2. intermittent stiction in intrafusal fibres is essential for postural stability,
3. postural plasticity may co-exist with postural rigidity,
4. permanent changes in maintained elbow joint angle may be obtained after passive movements imposed on the forearm.

These conclusions will all be discussed briefly.

6.1. THERE IS MORE TO POSTURE MAINTENANCE THAN THE REFLEXES OCCURRING IN RESPONSE TO A PERTURBATION

By studying EMG activities in response to imposed perturbations, it is easily forgotten that posture is also maintained without it being perturbed: when, for instance, sitting or standing, postural mechanisms control the angle of many joints simultaneously. This thesis demonstrates that a stable posture of the elbow joint is based on the co-contraction of antagonistic muscles, resulting in a local minimum in an energy landscape in which the forearm lies.

This antagonistic co-contraction is generated without us being aware: subjects were surprised to see how the EMG activity of their mm. biceps and triceps changed depending on whether a support of the forearm was provided or not. Without having the EMG activity displayed, one hardly notices the changes in muscle activation that occur when, for instance, the forearm is rested on the surface of a table.

6.2. INTERMITTENT STICTION IN INTRAFUSAL FIBRES IS ESSENTIAL FOR POSTURAL STABILITY

Hammond et al. (1956) already suggest that feedback control of muscle length, provided by the myotatic reflex, is of great advantage to a system controlling joint posture. In experimental

physiology, signs of the effectiveness of this feedback regulation are often investigated by imposing relatively large perturbations to a limb. This, however, does not provide the results expected: the gain of the stretch reflex is reported too small to correct for an imposed load (Matthews, 1966); and long latency stretch responses seem far more important for the restoration of joint posture than those at monosynaptic latency (Marsden et al., 1981; Nashner, 1976).

Some authors (e.g. Evarts, 1985) still follow Matthews (1966) in his conclusion that the gain of the myotatic stretch reflex is too small to correct adequately for changes in muscle load. However, it is noteworthy, that Matthews performs his calculations based on a spindle sensitivity of 10 pps/mm soleus stretch. Within the linear range, muscle spindles, however, are capable of displaying a much higher sensitivity to minute changes in muscle length (up to 280 pps/mm: Houk and Hasan, 1976). Consequently, the 30 g wt. (which is less than 2 % of the maximal isometric force to be generated by the cat soleus) per mm. soleus stretch that Matthews calculates may, in fact, be underestimated by a factor of 28[1]. Provided that the spindle remains in its linear range, the actual gain of the myotatic stretch reflex may, therefore, amount 840 g. wt. per mm. soleus stretch, which could certainly provide an important contribution to the postural mechanisms holding up the cat hindquarters against gravity.

The high spindle sensitivity within the linear range possibly depends upon stable cross-bridges within the intrafusal fibres (or: intrafusal stiction; Proske and Stuart, 1985). These stable actin-myosin bonds may become established at any spindle (or muscle) length, provided that the length is kept constant for some time (Gregory et al., 1990). Furthermore, it is shown that the spindle's high sensitivity only holds up to 100 μm soleus (or 25 μm spindle) stretch. Presumably, by a limited bending of the myosin heads, a larger stretch causes the yield of stable actin-myosin bonds.

So, the low synaptic gain of the monosynaptic stretch reflex may be compensated by the high length sensitivity muscle spindles display within the linear range. It is, therefore, again suggested that the myotatic stretch reflex is of major importance for the maintenance of elbow joint posture. Its contribution is even one of great subtlety as it makes use of the great adaptive capacities of muscle spindle sensitivity: the high gain with which the stretch reflex controls a stable limb posture, disappears instantaneously as soon as a (passive or active) limb movement causes the intrafusal stiction to yield.

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[1] In a later paper Matthews (1981) comes back on his original calculation and emphasizes the importance of this non-linearity in the spindle's length sensitivity.

When viewed in this way, the physiological action of the monosynaptic stretch reflex (preventing slow postural drift) may well escape from our attention, since it generates slight changes in muscle activation in response to minute changes in joint angle. In addition, these changes in muscle activation may occur over periods of time exceeding several hundreds of milli-seconds. They are hard to discern in a muscle's EMG, which is no more than a high frequency vector (or, carrier wave) of low frequency information.

The model simulation of the elbow joint (chapter 5) indicates that the high spindle sensitivity may also facilitate the problem of finding the right balance of muscle activations required for a certain joint posture. This relation between muscle activations and joint angle can hardly be expected a linear one, since there are many non-linearities even at a simple joint as the elbow. For instance, non-linearities arise from changing muscle working arms with changing joint angles, from non-linearities in the muscle's force-length and force-velocity characteristics, and because the human elbow joint is not perfectly cylindrical.
Some investigators have, therefore, concluded that the CNS has to dispose of a detailed image of all non-linearities arising for different reasons at a joint and uses this information to 'calculate' the appropriate muscle activations. The CNS is said to perform calculations on inverse dynamics. Others have suggested that the relation between joint posture and muscle activation is stored in a large lookup table (for a review see Hogan and Winters, 1990).
However, the adaptive capacity held with the muscle spindle sensitivity may largely facilitate the task of such complicated computational (or look-up) systems. The re-establishment of high spindle sensistivity ('intermittent stiction') at any given muscle length, may automatically ensure that the limb will move no further once it is adequately positioned at a desired joint angle. Due to the high spindle sensistivity for small movements, the gain of the myotatic reflex system is high enough to hold the arm in position, provided that the spindles remain in their linear range. Therefore, it is not necessary to assume that the CNS performs delicate computations in order to stabilize a given joint angle. Only the planning of muscle activations for the dynamic phases of a movement may still require calculations on inverse dynamics.

6.3. POSTURAL PLASTICITY MAY CO-EXIST WITH POSTURAL RIGIDITY

In chapter 4 it is shown that, in response to an imposed movement of the arm, early changes in EMG activity occur which, apparently, resist against a change of arm posture. Late EMG

responses, on the contrary, are found which seem to support and facilitate a change of elbow angle. These responses (the lengthening and shortening response) provide the elbow joint with postural plasticity: the capacity to assume a different joint angle.

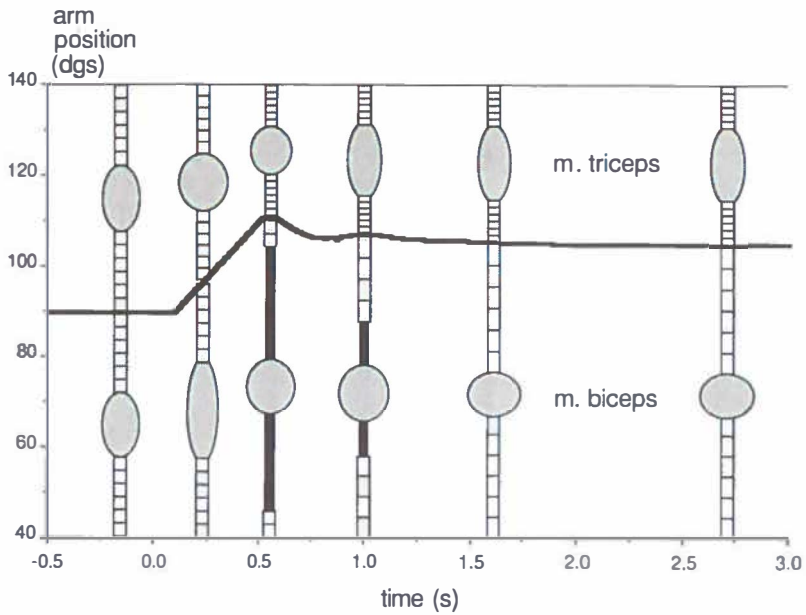

Fig. 6.1.

It is argued that postural plasticity is as important for the organism as postural stability: not only one single joint angle but many different ones need to be maintained. EMG recordings displayed in chapter 4 do not indicate what are the neural or peripheral mechanisms responsible for the lengthening and shortening response. Can intermittent stiction on its own explain for the observed changes in EMG activity, or are they caused by the action of specific neural pathways?

The active contraction of a muscle during passive shortening (Chapter 4) suggests that postural plasticity is controlled by the CNS. Possibly, the shortening response is caused by a disinhibition of alpha motoneurons, arising from a drop in Ib or II afferent activity (as result of decreasing muscle tension) or, caused by segmental interneuron systems giving rise to

reciprocal Ia inhibition or Renshaw inhibition. Ib or II afferents have been reported to provide (polysynaptic) inhibition to autogenetic alpha motoneurons: the Ib-afferents, which originate from Golgi tendon receptors, by Watt et al.(1976); and the secondary spindle afferents by Harrison and Jankowska (1985). Although it is also shown that II-afferents exert an excitatory action upon autogenetic alpha motorneurons (Matthews, 1969; Kirkwood and Sears, 1974; Stauffer et al. 1976) we used an inhibitory pathway from secondary afferents to autogenetic alpha motoneurons in a model simulation of postural plasticity at the human elbow joint (chapter 5). It is demonstrated that such an inhibitory pathway causes instability of the model's arm posture. This instability is compensated for by intrafusal stiction, which invokes a short-range postural rigidity.

However, the studies of Gregory et al. (1988, 1990) indirectly suggest that intrafusal stiction on its own may explain the observed postural plasticity at the elbow joint. Gregory et al. show how marked shifts in primary afferent sensitivity may occur dependent on past history of muscle stretch: when a muscle is made to contract isometrically at a small length and subsequently returned to an intermediate length, its spindle afferents fire at a frequency twice as large as when the muscle is made to contract at a large length before returning it to the same, intermediate, length.

Figure 6.1. demonstrates schematically, what possibly happens during a perturbation imposed on the forearm. An abrupt extension of the arm causes a dynamic burst of activity in the stretched m. biceps. Also, as the extension is large, it causes a yield of intrafusal stiction within the biceps spindles. Simultaneously, the muscle spindles in the shortening m. triceps follow the decreasing muscle length (possibly, because of a tonic level of fusimotor drive). Only when, after the perturbation, the forearm reaches its maximal amplitude and starts moving in opposite direction again, does the EMG of the triceps respond with a vigorous burst of activity, which decelerates the forearm.

In the mean time, the intrafusal stiction in the spindles of the m. biceps has partly re-established, but now at a greater muscle length. Therefore, the primary afferent discharge during shortening will be lower at identical muscle lengths than during lengthening. Whereas the m. biceps responds with a burst of EMG activity during lengthening it rapidly silences during shortening. This will cause a decrease in the muscle force pulling the arm back to its starting position.

Consequently, by inducing a dynamic yield and rebuild of intrafusal stiction, a passive movement of the arm may cause a permanent shift in joint posture.

6.4. PERMANENT CHANGES IN MAINTAINED ELBOW JOINT ANGLE MAY BE OBTAINED AFTER PASSIVE MOVEMENTS IMPOSED ON THE FOREARM

All perturbations used in chapters 3 and 4 had in common that they were elicited by imposing a brief flexing or extending torque on the forearm. So, just a pulse in torque may cause a permanent change in joint angle.

This observation may be extrapolated to the execution of active movements, generated by a subject's own muscle activity. It is deduced from the responses to a passive movement of the forearm, that for an active change in joint angle only a small agonist burst may be required: adaptive mechanisms in the proprioceptive feedback (e.g. muscle spindles) will ensure that a once acquired posture will be stabilized automatically.

Of course, the trajectory of the arm in response to such a short burst of agonist activity is not one of great movement control. This is the common explanation given for the occurrence of the so-called tri-phasic pattern of muscle activation observed when subjects make rapid (ballistic) arm movements (e.g. Brown and Cooke, 1990): A first agonist burst accelerates the arm (AG1); then a second antagonist burst, causes a deceleration (ANT1); followed by activation of the agonist again, presumingly, in order to stabilize the limb after movement termination (AG2).

In response to a passive movement of the arm, however, two bursts of activity may be seen in the agonist and antagonist which correspond in timing with the latter two bursts observed during an active movement of the arm. In response to a passive movement of the forearm, the stretched muscle responds with a burst of activity, possibly corresponding with ANT1. And, somewhat later, the shortening muscle displays a burst of EMG activity, attributed to a shortening response but possibly corresponding with AG2 during an active movement (Angel, 1983).

The comparison of phasic EMG responses during passive movements with those observed during active movements might elucidate what are the voluntary and what the automatic components in the execution of a ballistic movement.

CHAPTER 7

Summary

This thesis deals with some questions on posture maintenance at the human elbow joint.
It is generally believed that posture is maintained by the action of myotatic reflexes. A myotatic (or stretch) reflex is the involuntary muscle contraction that occurs in response to a brief stretch[1] of that muscle. The reflex is generated by the activation of muscle spindles within the stretched muscle. These receptors generate action potentials which travel along primary afferent fibre into the spinal cord and elicit a reflex excitation of the autogenetic alpha motoneurons (the neurons in the spinal cord which innervate the same, stretched, muscle). So a small change in the angle of the elbow joint may, depending on its direction, stretch either the elbow extensors or its flexors and evoke a reflex muscle contraction which appartently aims at restoring the original posture of the joint.
However, as explained in chapter 1, such a view on posture control suggests a rigid maintenance of one single joint posture. The system can only escape from this rigidity if, for instance, the central nervous system (CNS) makes use of its capacity to alter the muscle spindles' sensitivity by gamma modulation. This thesis describes a number of experiments which were designed based on the idea that gamma motorneurons are inhibited by muscle spindle II-afferents. It was expected that such a feedback mechanism might prevent the elbow joint from becoming rigid. That such a neural pathway might exist, was supported by evidence provided by a series of investigations (Hunt, 1951; Eldred, Granit and Merton, 1953; Fromm, Haase and Noth, 1974; Ellaway and Trott, 1978). However, in the course of this study the starting hypothesis had to be withdrawn and replaced by another.
How is the muscle spindle sensitivity changed by a change in fusimotor activity? Matthews (1981) describes an hypothesis how the differences in primary afferent sensitivity can be explained out of differences in the contractile properties of the dynamic bag_1 on the one hand, and of the static bag_2 and nuclear chain fibres on the other: he suggests that during dynamic

[1] The tendon jerk of the knee is the most prominent example of a stretch reflex: the muscle stretch evoked in the m. quadriceps by a tap on its tendon with a tendon hammer, causes a brief contraction of this muscle and, thereby, a jerking movement of the lower leg.

activation of the bag_1 fibre it is mainly the viscosity[2] of the intrafusal fibre which increases, whereas static fusimotor activation of the bag_2 and nuclear chain fibres is though to increase the fibers' elasticity[3]. We developed a muscle spindle model which was based on this idea (chapter 2). It proved possible to model a muscle spindle's primary afferent response out of a simulation of intrafusal mechanical events.

Another important characteristic of the spindle's primary afferent response is its high sensitivity to minute changes in muscle length (Hasan and Houk, 1971). This high length sensitivity is of a limited range (up to 25 μm spindle stretch) and is thought to arise from stable actin myosin bonds (usually referred to as intrafusal stiction) in the contractile parts of the dynamic bag_1 fibre. This short range stiffness in the intrafusal fibres causes a stretch at the spindle poles to be transmitted directly to its sensory region. Thereby, the spindle attains such a high length senstivity. This mechanism was also incorporated in the spindle model.

If the idea were true that gamma modulation prevented postural rigidity, as forwarded in chapter 1, this would also mean that signs should be found indicating postural plasticity (the capacity to maintain any joint posture within the limits of limb flexibility). The heart of the investigation was a simple observation at the human elbow joint indicating that postural plasticity could indeed be observed: when instructing a subject to hold up his arm in 90 degrees abduction at the shoulder and 90 degrees flexion at the elbow joint, it was observed that upon small, abrupt perturbations of the limb, for instance by a tap with a tendon hammer, the arm would return accurately to its original position, whereas upon large and prolonged perturbations the arm gave in to the movement and assumed a new posture, shifted in the direction of the perturbation.

Chapter 3 and 4 describe an experimental set-up for the controlled investigation of this observation. Chapter 3 investigates the characteristics of posture maintenance at the human elbow during the first 100 ms of a perturbation. By taking only this limited period of time, the effect of possible reflex action was excluded. Under these circumstances, it was demonstrated that the posture of the elbow is maintained by the mechanical impedance of the joint. The mechanical impedance of a joint is its resistance against a perturbation due to the moment of inertia of the moving limb (in this case the forearm) and due to visco-elastic properties of its flexors (mm. biceps and brachialis) and extensor (m. triceps). The relatively large moment of inertia of the forearm, prevents it from spontaneously starting to move at high velocity. Movements at a slow velocity (or the so-called slow postural drift) are less well antagonized.

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[2] The viscosity of a muscle is the force with which a muscle resists a change in length.

[3] The elasticity of a muscle is the force it exerts at a constant length.

The prevention of slow drift depends entirely upon the joint stiffness generated by a co-contraction of the muscles governing the elbow joint. It is suggested that the balanced tuning of antagonistic muscle activity required for a stable joint posture is derived from the high muscle spindle sensitivity occuring as result of intrafusal stiction.

Chapter 4 investigates the long term effects of a perturbation imposed on the forearm. It demonstrates that, during the early phases of a perturbation, EMG responses can be recorded which, apparently, oppose the perturbation, whereas, at a later stage, changes are observed which facilitate the passive movement of the arm and allow the elbow to assume a new joint posture. The latter responses provide the elbow joint with postural plasticity.

A study of the final angle at which the elbow joint settled after perturbations of various amplitudes, demonstrated differences in response to small versus large perturbations. After a small perturbation, the forearm returns to its original position. This re-positioning is suggested to arise from the action of the myotatic reflex, as its high sensitivity due to intrafusal stiction remains undisturbed. After a large perturbation (more than 2 degrees; chapter 3), the intrafusal stiction is expected to yield, resulting in a drop in spindle sensitivity. Plastic responses take over and allow the arm to stabilize at a new and different joint angle (at which the intrafusal stiction can re-establish).

This explanation on the physiological processes taking place during a perturbation of the arm was further elaborated by a computer model. Chapter 5 describes a model of the human arm. It consists of a mass (the forearm), rotating around a cylindrical joint (the elbow) and held in place by the flexors and extensor of the joint. This model incorporated, with slight modifications, the muscle spindle model of Chapter 2: it proved very important for the model's behaviour to allow intrafusal stiction that had yielded at one stage of a simulation to re-establish at a later. In the model, this so-called intermittent stiction proved essential for a stabilization of the elbow joint posture: its incorporation prevented the model's elbow joint from displaying slow postural drift.

In Chapter 6 it is concluded that postural plasticity may co-exist with postural rigidity without rendering unstable the posture of the elbow joint. It is also suggested that the adaptive properties incorporated within the muscle spindles of antagonistic muscle groups may facilitate calculations on inverse dynamics assumed to occur within the CNS. (Calculations on inverse dynamics provide the muscle activations required to move a limb or hold it in position, taking into account all non-linearities arising from changing muscle working arms, non-linear force-length and force-velocity relationships, etc.)

SAMENVATTING

Dit proefschrift gaat over houdingsregulering van de elleboog. Twee aspecten worden met name belicht: plasticiteit en rigiditieit. Onder plasticiteit verstaat men de mogelijkheid om verschillende houdingen aan te nemen; onder rigiditeit de mogelijkheid om zekere houdingen te bewaren.

In het algemeen wordt verondersteld dat aan het in stand houden van houdingen spierrekkingsreflexen ten grondslag liggen. Een spierrekkingsreflex (myotatische reflex) is de onwillekeurige samentrekking van een spier als antwoord op een kortdurende rek van die spier[1]. De reflex ontstaat door de activering van spierspoelen[2] die in de gerekte spier liggen. Bij

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[1] Een goed voorbeeld van een spierrekkingsreflex is de kniepeesreflex: een tik met een peeshamer op de pees van de kniestrekker in het bovenbeen rekt de spier die, ten gevolge van de spierrekkingsreflex, contraheert waardoor het onderbeen opzwaait.

[2] Spierspoelen zijn complexe intramusculaire receptororgaantjes, die als lengteopnemer voor de spier fungeren. Zij bestaan uit gemodificeerd spierweefsel dat nog wel zijn contractiliteit heeft behouden. Spierspoelen liggen door de hele spier verspreid. Om spierspoelvezels van overige spiervezels te onderscheiden spreekt men van intrafusale en extrafusale vezels. Men onderscheidt twee soorten intrafusale spiervezels (Lat. fusus: spoel): nuclear-bag (letterlijk: kernzak) en de nuclear-chain (letterlijk: kernstreng) vezels. Er zijn twee types nuclear-bag vezels: de dynamische bag_1 en de statische bag_2 vezel. Iedere nuclear-bag vezel bestaat uit een centraal gedeelte met daarin een groot aantal kernen. Dit gedeelte is niet contractiel. Aan weerszijden van dit centrale gedeelte bevinden zich de zogenaamde polaire gedeeltes (Eng. polar regions), die actine en myosine filamenten bevatten en derhalve hun contractiele eigenschappen hebben behouden. In een nuclear chain vezel liggen de kernen verspreid over de gehele spiervezel; ook de contractiele filamenten worden over de gehele vezellengte aangetroffen. Op de omslag van dit proefschrift is een foto afgebeeld van een spierspoel met daarin een nuclear chain en een nuclear bag vezel. Preparaat en foto zijn vervaardigd door dr. R.S.B. Liem, ook van de vakgroep Neurobiologie en Orale Fysiologie.
Een spierspoel zendt zijn informatie naar het centrale zenuwstelsel (CZS) toe via afferente zenuwvezels. Er zijn twee types afferenten: de primaire (of Ia-) afferenten, en de secundaire (of II-) afferenten. Het receptieve gedeelte van een Ia vezel wordt "annulospiral ending" genoemd, dat van een II vezel "flower spray ending". Zowel de Ia- als de II-afferenten geven informatie over de vigerende spierlengte (statische gevoeligheid). Ia-afferenten geven ook informatie over de snelheid waarmee een spier van lengte verandert (dyamische gevoeligheid).
De intrafusale spiervezels staan onder invloed van het CZS via efferenten die afkomstig zijn van gamma motorneuronen in het ruggemerg . Gamma (ook wel fusi-) motoneuronen worden, afhankelijk van hun effect op de spierspoel, ingedeeld in twee types: (1) statische fusimotoire neuronen (γ_s) die de lengtegevoeligheid en (2) dynamische fusimotoire neuronen (γ_d) die de snelheidsgevoeligheid van de spoel verhogen. Registratie van het gedrag van spierspoelen onder microscopische observatie (Boyd, 1985), laat zien dat de twee types fusimotoire neuronen verschillende types intrafusale vezel innerveren: de γ_s neuronen innerveren de

rek zenden deze spierspoelen zenuwimpulsen (actiepotentialen) naar het ruggemerg langs de zogenoemde primaire afferente zenuwvezels (Ia-afferenten). In het centrale zenuwstelsel aangekomen, veroorzaken deze actiepotentialen een toeneming in vuurfrequentie (excitatie) van de neuronen die de gerekte spier innerveren (de homologe alpha-motorneuronen). Men stelt zich de houdingsregulering van een lichaamsdeel, in ons geval de onderarm, als volgt voor: wanneer er, ten gevolge van een verstoring, een kleine verandering in de gewrichtshoek van het elleboogsgewricht onstaat, worden, afhankelijk van de richting van de verstoring, de buig- dan wel de strekspieren van het gewricht gerekt. In de gerekte spier ontstaat een reflexmatige contractie (spierrekkingsreflex) die erop gericht lijkt om de oorspronkelijke gewrichtshoek te herstellen.
Zoals in Hoofdstuk 1 wordt betoogd is dit een veel te eenvoudige voorstelling van zaken. Immers, als houdingsregulering van de onderarm alleen op spierrekkingreflexen zou zijn gebaseerd dan zou het elleboogsgewricht slechts één gewrichtsstand kennen; het zou rigide zijn. Een mogelijkheid voor het centrale zenuwstelsel (CZS) om aan deze rigiditeit te ontsnappen is, om door modulering van het gammasyteem, de spierspoelgevoeligheid (als functie van de grootte en snelheid van de verstoring) te veranderen.
Dit proefschrift beschrijft een aantal experimenten die gebaseerd zijn op dit idee. Als eerste hypothese werd getoetst dat bij houdingsverstoringen boven een kritische grootte de gamma-motorneuronen van de spierspoelen in de gerekte spieren geïnhibeerd worden door hun eigen II-afferenten. Een dergelijk terugkoppelingssysteem zal theoretisch het onstaan van rigiditeit voorkomen. Steun voor deze hypothese werd in de literatuur gevonden bij Hunt (1951), Eldred, Granit en Merton (1953), Fromm, Haase en Noth (1974) en Ellaway en Trott (1978), alhoewel vanaf het begin vaststond dat ook veel literatuur tegen het bestaan van een dergelijk mechanisme pleitte (Appelberg ea., 1983; Hunt en Paintal, 1958; Hulliger ea., 1982).

Volgens Matthews (1981) wordt de gevoeligheid van een spierspoel bepaald door de verschillen in biomechanische eigenschappen van de samenstellende intrafusale spiervezels, en het verschil in hun gedrag onder de twee vormen van gamma-innervatie. Hij veronderstelt dat vooral de viscositeit[3] van de dynamische bag_1 onder invloed staat van het dynamische

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statische bag_2 en de nuclear chain vezels, terwijl de γ_d neuronen de dynamische bag_1 vezel innerveren.

[3] De viscositeit van een spier is de weerstand die een spier uitoefent tegen een *verandering* in lengte. Het is de dempende eigenschap van een spier.

fusimotoire systeem, terwijl de elasticiteit[4] van de statische bag_2 en nuclear chain vezels vooral wordt beinvloed door het statische fusimotoire systeem.
Met dit idee als uitgangspunt, hebben wij een computermodel van een vertebrate spierspoel opgesteld dat de vuurfrequentie van een primaire spierspoelafferent (Ia afferent) onder verschillende prikkelregimes simuleert. Het bleek mogelijk om de verschillen in gevoeligheid van een Ia-afferent voor dynamische en statische fusimotor condities met een grote mate van realisme te simuleren op grond van verschillen in de biomechanica van intrafusale spiervezels. Dit spierspoelmodel wordt in Hoofdstuk 2 beschreven.
Onder zekere omstandigheden hebben Ia afferenten een extreem hoge gevoeligheid voor zeer kleine lengteveranderingen (Hasan en Houk, 1971). Dit fenomeen wordt waarschijnlijk veroorzaakt doordat bij een kleine en snelle rek de bruggen tussen de actine en myosine filamenten van de polaire delen van de dynamische bag_1 vezels niet direct verbroken worden. Hierdoor gedragen de polaire delen zich als een soort klitteband (Eng. intrafusal stiction). Bij het begin van de rekking wordt (door het bestaan van bruggen tussen het actine en myosine) alle rek doorgegeven aan het centrale deel van de bag_1 vezel, waardoor de Ia afferent met hoge frequentie gaat vuren. Bij rek groter dan ca. 25 μm verbreken de actine/myosinebruggen en de vuurfrequentie van de Ia afferent neemt plotseling af.
Introductie van het fenomeen "stiction" in het spierspoelmodel bleek essentiëel te zijn voor het simuleren van een realistisch vuurgedrag van zijn Ia afferent.

Ons onderzoek naar plasticiteit en rigiditeit van de onderarm is gebaseerd op de volgende simpele observatie. Wanneer men de arm ophoudt met een abductie van negentig graden van de schouder, en een buiging van de elleboog van eveneens negentig graden, blijkt bij een kleine verstoring de onderarm terug te keren naar zijn uitgangspositie; wanneer de arm over een grote hoek wordt bewogen neemt hij daarentegen een nieuwe stand in.
Hoofdstuk 3 en 4 beschrijven hoe dit verschijnsel onder gecontroleerde omstandigheden werd bestudeerd.
Hoofdstuk 3 beschrijft het gedrag van de onderarm gedurende de eerste 100 ms van een verstoring. Binnen deze tijd hebben eventueel opgewekte reflexen nog geen mechanisch effect op de positie van de arm; dit komt door de grootte van de looptijden in de reflexkring en de tijdconstanten die aan de excitatie/contractie mechanismen van de armspieren ten grondslag

[4] De elasticiteit van een spier is de kracht die een spier uitoefent bij een *zekere* lengte. Het is de elastische eigenschap van een spier.

liggen. In de eerste 100 ms blijkt de gewrichtshoek van de elleboog uitsluitend bepaald te worden door de zogenaamde mechanische impedantie van het systeem. Onder "mechanische impedantie" verstaat men de weerstand die de houdingspieren van de onderarm (en het elleboogsgewricht) uitoefenen tegen houdingsveranderingen. De grootte van deze mechanische impedantie wordt grotendeels bepaald door de massatraagheid van de onderarm en de dempende en elastische eigenschappen van de houdingsspieren en het gewricht. Het blijkt dat de grootte van de massatraagheid van de onderarm ervoor zorgt dat de arm niet spontaan, en met grote snelheid, gaat bewegen. Langzame houdingsveranderingen (Eng. slow drift) worden echter nauwelijks door de grootte van massatraagheid beïnvloed. Slow drift wordt voornamelijk tegengegaan door de "stijfheid" van het elleboogsgewricht (Eng. joint stiffness) dat wil zeggen door de mate van (gelijktijdige) samentrekking van onderarmbuigers en -strekkers. De precieze afregeling van de activiteit van beide spiergroepen is waarschijnlijk automatisch en gebaseerd op de extreem hoge gevoeligheid van spierspoelen voor kleine veranderingen in lengte.

Hoofdstuk 4 beschrijft de lange termijn effecten van een armverstoring. Deze verstoringen blijken -afhankelijk van de grootte - vroege en late reacties te initiëren in zowel de gerekte als in de verslappende spieren. De vroege responsies lijken erop gericht te zijn om de effecten van de verstoring teniet te doen, terwijl de late reacties het tegengestelde lijken te bewerkstelligen: ondersteuning van de beweging van de onderarm en consolidering in een nieuwe positie. Met andere woorden de late responsies maken het mogelijk dat de onderarm zich plastisch gedraagt en een andere gewrichtshoek aanneemt.
Er blijken grote interindividuele variatie's te bestaan. In het algemeen is de mate van plasticiteit van het elleboogsgewricht afhankelijk van de grootte van een verstoring; niet van de verstoringssnelheid. Na een verstoring met kleine amplitude gedraagt de arm zich rigide, na een verstoring met grote amplitude plastisch. Wij veronderstellen dat het wel of niet bestaan van stiction aan dit gedrag ten grondslag ligt.
Om dit idee verder uit te werken hebben wij een modelsimulering van de beschreven experimenten uitgevoerd, omdat de effecten van stiction op rigiditeit en plasticiteit van de onderarm niet experimenteel te meten zijn. Deze simulatie is in Hoofdstuk 5 beschreven.
Het model bestaat uit een massa (de onderarm), die roteert om een cylindrisch gewricht (de elleboog) en waarvan de positie bepaald wordt door een model van twee buig- (mm. biceps en brachialis) en één strekspier (m. triceps). In de spiermodellen zijn spierspoelmodellen (zoals beschreven in Hoofdstuk 2) opgenomen. Om realistische modelsimulaties van onze

onderzoeksresultaten te krijgen bleek het noodzakelijk een nieuwe spierspoeleigenschap te postuleren die wij "intermittent stiction" hebben genoemd. Onder intermittent stiction verstaan wij de eigenschap van een spierspoel bij constante lengte om actine/myosinebruggen in de dynamische bag_1 vezels te vormen wannneer de rekkracht onder een zekere drempel is gekomen; bij overschrijding van deze drempel worden de actine/myosinebruggen verbroken. Deze uitbreiding van het spierspoelmodel met intermittent stiction bleek noodzakelijk om slow drift van de onderarm te voorkomen.

Hoofdstuk 6 is een discussie over de mechanismen die mogelijk aan onze onderzoeksresultaten (rigiditeit van de onderarm kan samengaan met plasticiteit zonder dat het elleboogsgewricht instabiel wordt) ten grondslag liggen. Er wordt daarbij het argument aangevoerd dat intermittent stiction de taak van het CZS vergemakkelijkt bij het berekeningen van de inverse dynamica.[5]

[5] Men veronderstelt dat het CZS dergelijke berekeningen uitvoert om de spieractiveringspatronen te vinden voor het bewaren van een houding of het initiëren van een beweging. Bij deze berekingen zou het CZS rekening houden met alle mogelijke bronnen van niet-lineariteit (ten gevolge van moment-armen van spieren die veranderen met de gewrichtshoek en van de niet-lineare kracht-lengte en kracht-snelheidsrelaties van een spier).

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